The Journal of the American Osteopathic Association

JULY 1961 • VOLUME 60

Manipulative body mechanics therapy

A report to the membership (continued)

Table of contents on page A-2

because it
MAKES
ENEMAS OBSOLETE
IN
POSTPARTUM
CONSTIPATION
your Rx for

Senokot®*

TABLETS / GRANULES

means

A GRATEFUL PATIENT

SENOKOT drastically cuts down the use of enemas: Of 319 puerperal patients treated with SENOKOT Granules/Tablets, as reported in three separate studies,[1,2,3] only 5.6% required enemas...in contrast to controls, in whom enema requirements ranged from 83% to 87%.

Virtually eliminates side effects: The total incidence of side effects in the above three studies was limited to 2.5%—out of 319 patients, only 8 reported some loose stools or griping.[2] No reactions were observed in the other two series. "A remarkable finding was the complete absence of side effects," comment Suarez and co-workers.[1]

Helps to restore normal bowel activity—before and after delivery: By the fourth postpartum day, "practically all treated patients had resumed normal bowel activity."[1] Restoration of normal bowel function was also seen in 97% of 59 antepartum patients included in one of the studies.[3] SENOKOT acts *physiologically*, inducing normal, effortless evacuation through *neurostimulation of the myenteric plexus of the colon*... not by unreliable bulk or contact irritation.

Obstetrical patients are grateful for SENOKOT: All three studies note the outstanding patient acceptance: "Patient acceptance was highly favorable..."[1] ..."The flavor...was entirely acceptable and pleasing to these 100 women."[2] ..."Acceptance of the product was uniformly favorable."[3] Because it eliminates the discomfort of enemas, rapidly restores normal bowel function, and is virtually free of side effects, your patient will be grateful when you prescribe SENOKOT Granules/Tablets.

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Supply: 16, 8 and 4 ounce canisters: Bottles of 30 and 100 tablets.

References: 1. Suarez, J. et al.: Internat. Rec. Med. *173*:639 (Oct.) 1960. 2. Kasdon, S. C., Morentin, B. O.: J. Internat. Coll. Surg. *31*:455 (Apr.) 1959. 3. Wager, H. P., Melosh, W. D.: Quart. Rev. Surg. Obst. & Gynec. *15*:30 (March) 1958.

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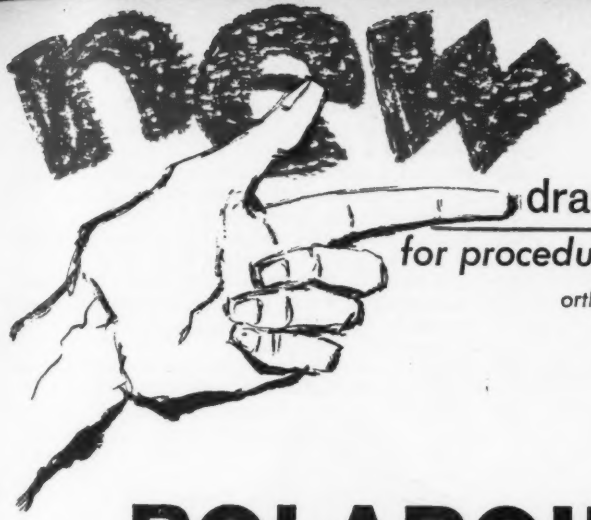

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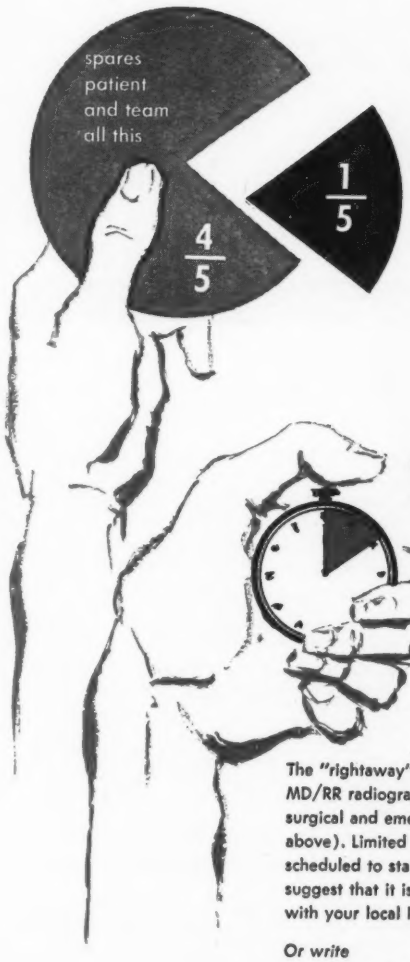

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July 1961, Volume 60, Number 11

887 Manipulative body mechanics therapy: II. Upper dorsal lesion complex. *J. Willard Johnson, D.O., Minneapolis, Minnesota*

896 Prevention and minimization of barotraumatic injuries associated with scuba diving. *Robert W. Shelby, D.O., St. Louis Missouri*

902 Juvenile rheumatoid arthritis (Still's disease). *Louis F. Amalfitano, D.O., Highland Park, Michigan*

907 The diagnosis of pulmonary embolism and infarction: I. Clinical and electrocardiographic findings. *Alexander Price, D.O., and Murray H. Cohen, D.O., Philadelphia, Pennsylvania*

917 Preparation and premedication for the first stage of labor. *Roy L. Fischer, B.S., D.O., Dallas, Texas*

920 Pulmonary roentgen changes following cholecystectomy and their relationship to respiratory morbidity. *Milton Metzman, D.O., Philadelphia, Pennsylvania*

927 Pruritus ani: Etiology and therapy. *Richard H. Hurst, D.O., F.A.O.C.Pr., Denver, Colorado*

935 Dynamic concept of psychopathology. *Martin B. Goldstein, B.S., D.O., Philadelphia, Pennsylvania*

939 Editorials

943 Activities of the American Osteopathic Association

953 Product News

961 Current Literature

965 Book Reviews

A- 73 Conventions and Meetings

A- 75 State and National Boards

A-138 Books Received

A-153 Changes of Address

A-168 Applications for Membership

INFORMATION FOR CONTRIBUTORS

THE JOURNAL OF THE AMERICAN OSTEOPATHIC ASSOCIATION is the official scientific publication of the American Osteopathic Association. Articles are accepted with the understanding that they have not been published or accepted for publication elsewhere.

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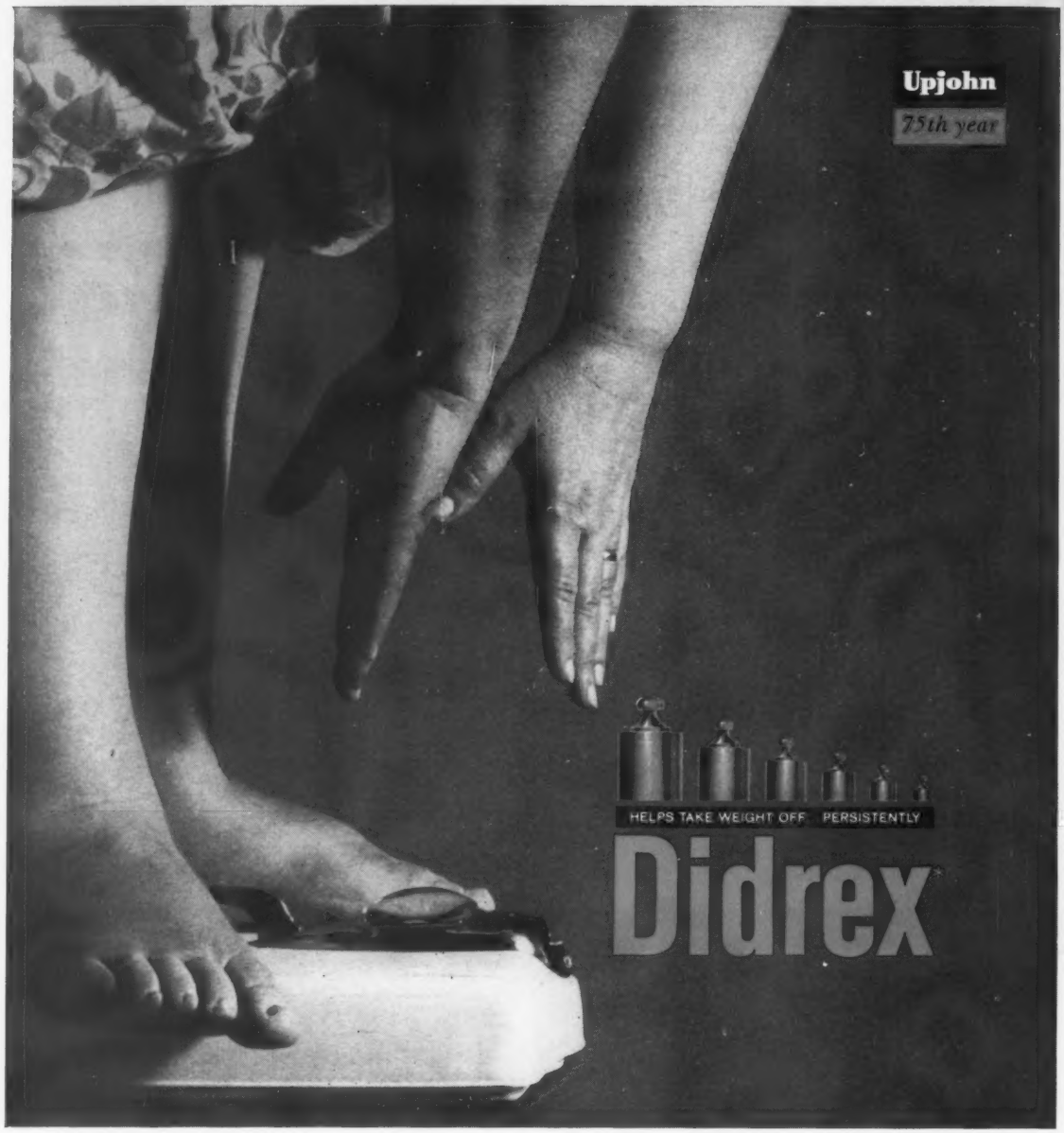

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Contraindications: None known to date. However, use with caution in moderate or severe hypertension, thyrotoxicosis, acute coronary disease, or cardiac decompensation.

Dosage: Initiate appetite control with ½ to 1 tablet (25 to 50 mg.) in mid-morning or mid-afternoon, according to the patient's eating habits for several days. Then "adjust" dosage to suit each patient's needs to a maximum of 3 tablets daily (150 mg.).

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Supplied: 50 mg., benzphetamine hydrochloride, press-coated, scored tablets, in bottles of 100 and 500.

*Trademark—brand of benzphetamine hydrochloride, Upjohn.

References: 1. Stough, A. R.: Weight loss without diet worry: use of benzphetamine hydrochloride (Didrex). Journal of the Oklahoma State Medical Association, 53:760-767 (November) 1960. 2. Oster, H., and Medlar, R.: A clinical pharmacologic study of benzphetamine (Didrex), a new appetite suppressant. Arizona Medicine, 17:398-404 (July) 1960. 3. Simkin, B., and Wallace, L.: A controlled clinical trial of benzphetamine (Didrex). Current Therapeutic Research, 2:33-38 (February) 1960.

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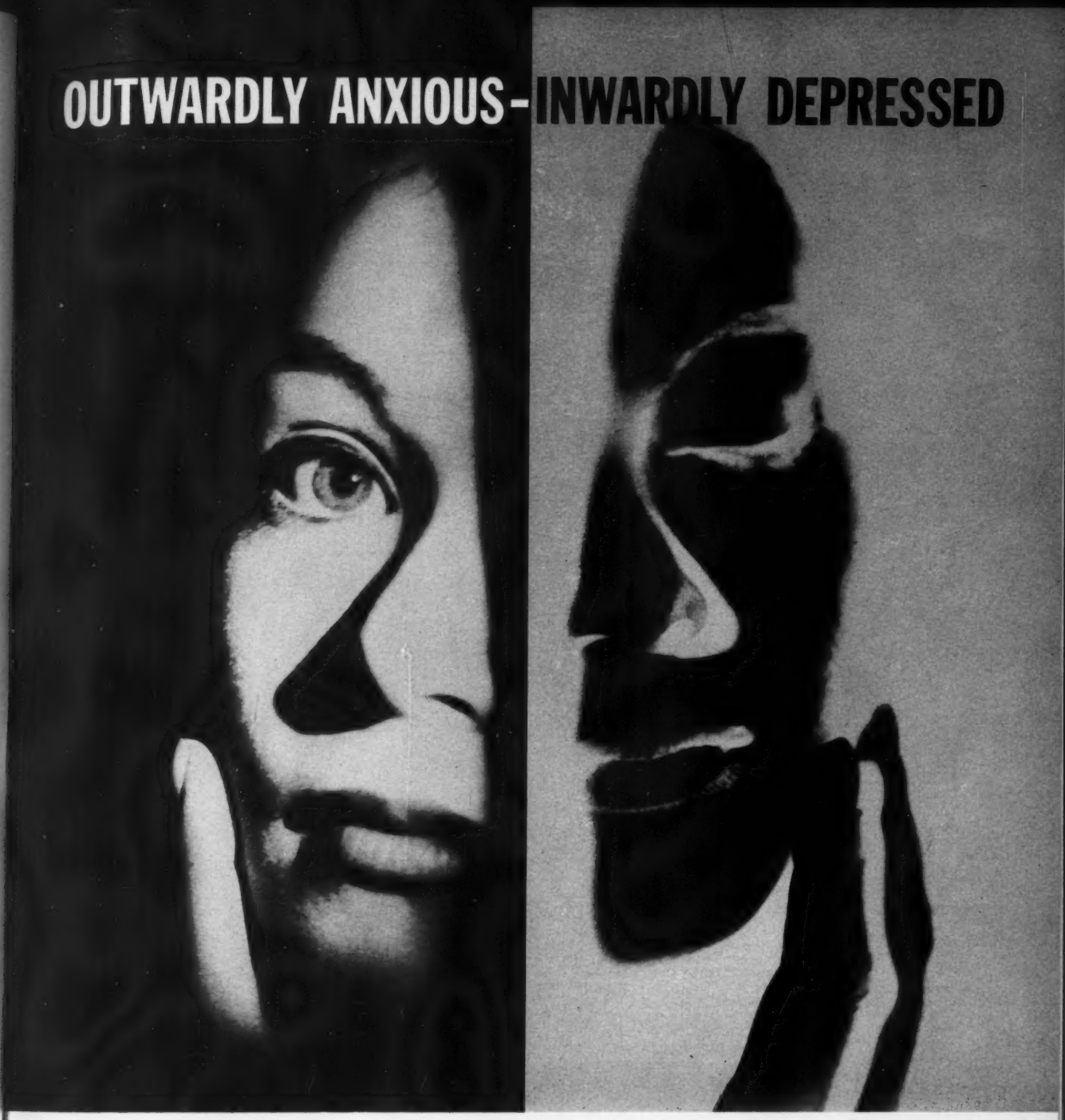

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*Hobbs, L. F.: VIRGINIA M. MONTH. *86*:692, (December) 1959.

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* Blood, 15:540 (April) 1960.

** Am. Pract. & Digest Treat., 10:461 (March) 1959.

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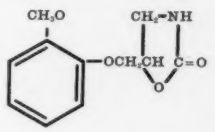

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References: (1) Ratowsky, S., and Padernacht, E. D.: Clin. Med. *8*:512-514 (March) 1961. (2) Voulgaris, D. M.: Dysmenorrhea: Cramps or Psyche?, Scientific Exhibit, Am. Acad. G. P, Philadelphia, March 21-24, 1960. (3) Voulgaris, D. M.: Obst. & Gynec. *15*:220-222 (Feb.) 1960.

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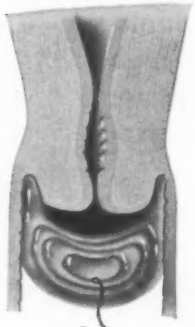

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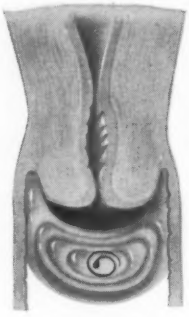

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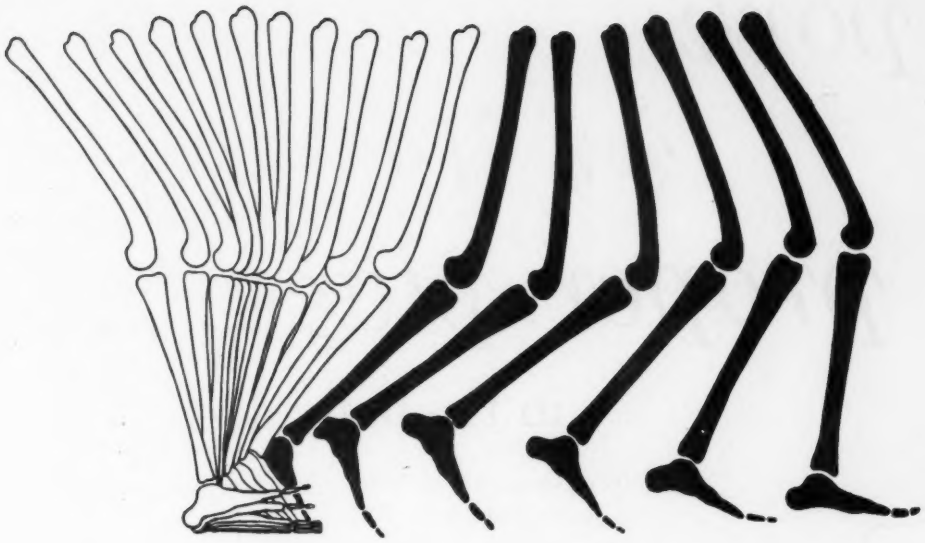

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For injection into the tendon sheath, 4 to 30 mg. is a usual range (in recurrent or chronic conditions, repeat injections may be needed).

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20 mg. per cc.
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Polyethylene glycol 4000	29.6 mg.
Sodium chloride	8.9 mg.
Myristyl-gamma-picolinium chloride	0.19 mg.
Water for injection	q.s.

Supplied: 5 cc. vials

1. Norcross, B. M., and Winter, J. A.: Methylprednisolone acetate: a single preparation suitable for both intra-articular and systemic use, New York J. Med. *61*:552 (Feb. 15) 1961.

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1. Leverton, R.M., and Odell, G.V.: The Nutritive Value of Cooked Meat, Oklahoma Agricultural Experiment Station, Oklahoma State University, Miscellaneous Publication MP-49, 1958.

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Two Pyridium tablets t.i.d. relieve the pain of urinary infection in only 30 minutes. During the first 3 to 4 days of therapy, Pyridium, prescribed along with any antibacterial of your choice, will make your patient comfortable until the antibacterial reduces inflammation and controls the infection.

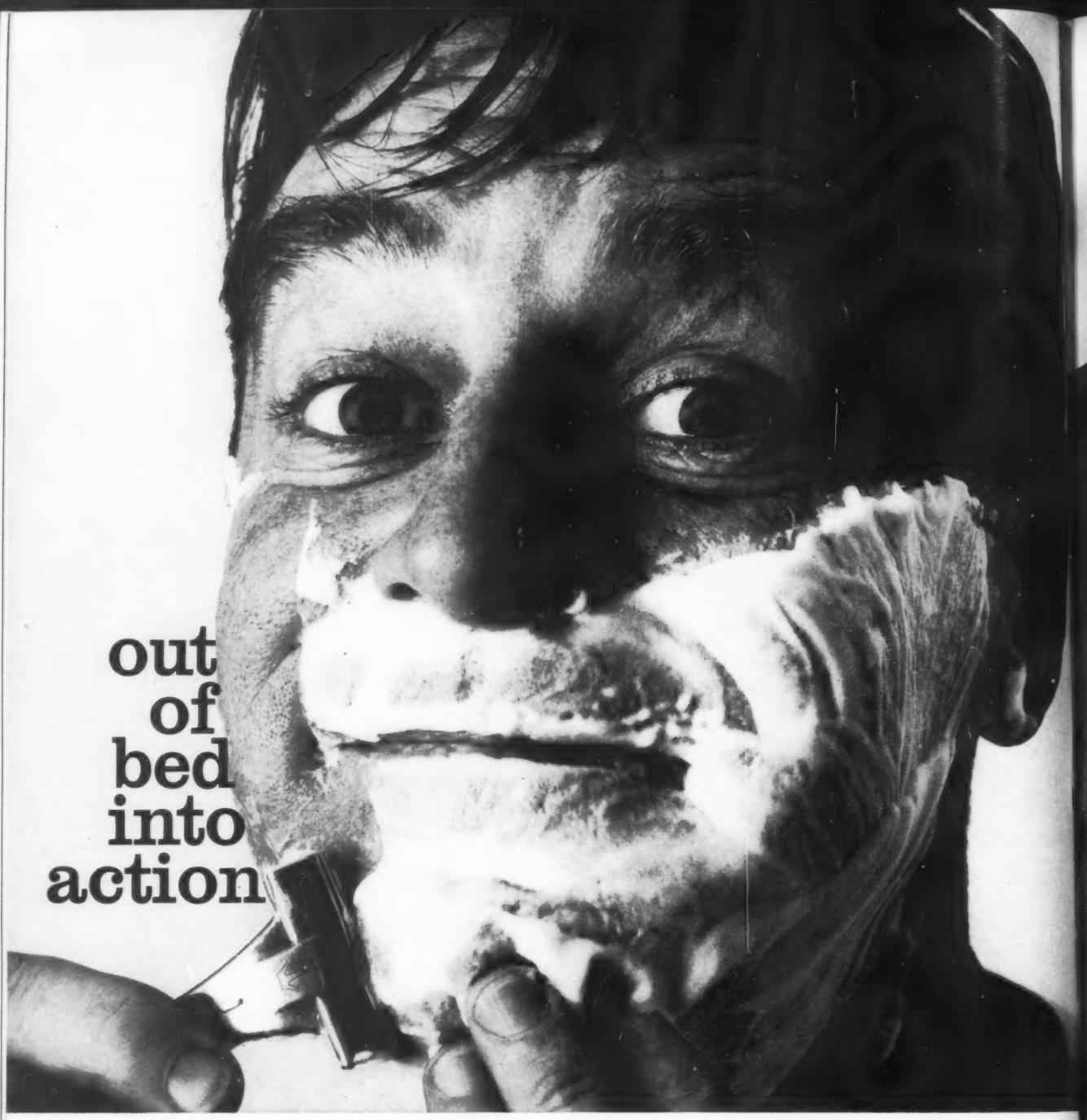
AVERAGE DOSE: Adults—2 tablets t.i.d. Children 9 to 12 —1 tablet t.i.d. SUPPLIED: 0.1 Gm. tablets, bottles of 50. PRECAUTIONS: Pyridium is contraindicated in patients with renal insufficiency and/or severe hepatitis. *Full dosage information, available on request, should be consulted before initiating therapy.*

WARNER CHILCOTT

MORRIS PLAINS, N.J.

6P12

Reg. U.S. Pat. Off. TEDRAL GELUSIL PROLOID PERITRATE NANDELAMINE



After a night of deep, refreshing sleep—this is the promise of Noludar 300. One capsule at bedtime acts quickly... eases your patient into sleep without pre-excitement, gives up to 6 or 8 hours of undisturbed sleep without risk of habituation, without toxicity or even minor side effects. Try Noludar 300 for your next patient with a sleep problem. Chances are he'll tell you

"I slept like a log"

NOLUDAR® 300

brand of methyprylon

300-mg capsules

ROCHE LABORATORIES • Division of Hoffmann-La Roche Inc • Nutley 10, New Jersey

WHY ACID MANTLE?

A DISTINCT THERAPEUTIC ENTITY*
Restores and maintains skin's normal protective acidity—
speeds natural healing and helps sensitive skin resist irritants and infection.

A NOTABLE VEHICLE
Special water-miscible, evaporable base assures better dispersion,
greater concentration of active ingredients in contact with skin—
increases response through its own therapeutic action.

*Supplied: in Creme and Lotion (pH 4.6).

now low-cost steroid topicals help insure sustained improvement without setbacks

new 1/8% Cort-Dome®

Creme · Lotion (pH 4.6)

micro-dispersed hydrocortisone alconol in Acid Mantle® for economical long-term therapy

IMPROVED PROCESS PERMITS EFFICIENT DOSAGE REDUCTION Once the dermatosis is brought under control with higher steroid concentrations, new ⅛% Cort-Dome, potentiated by the *micro-dispersion* of hydrocortisone in its Acid Mantle vehicle, can help insure therapeutic success—with less likelihood of flare-ups—until the skin's natural defenses are restored. The markedly lower cost of ⅛% Cort-Dome adds assurance that the patient will continue therapy as directed.

CORT-DOME—Tailored Steroid Topicals For individualized therapy and unique versatility in control of dermatologic problems at reasonable cost, Cort-Dome is supplied in a wide choice of concentrations: 2%, 1%, and ½% to initiate therapy; ¼% and new ⅛% for maintenance therapy. When infection is a consideration, Neo-Cort-Dome® provides neomycin sulfate 5 mg./Gm. in the same formulations at no extra cost.

WORLD LEADER IN DERMATOLOGICALS
DOME CHEMICALS INC.
New York 23, New York

01261

'THORAZINE' PRESCRIBING INFORMATION

Because of its pronounced calming effect, 'Thorazine' is an outstanding agent for patients in whom agitation and hyperactivity present a management problem. In severe cases, initial use of intramuscular administration may be desirable to control symptoms promptly.

Before prescribing 'Thorazine' for other indications than those given below, the physician should be familiar with the dosage, side effects, cautions and contraindications for such uses. This information is available in the *Thorazine® Reference Manual* and *Physicians' Desk Reference*, and from your SK&F representative or your pharmacist.

ADULT DOSAGE AND ADMINISTRATION

Dosage should always be adjusted to the response of the individual and the severity of the condition. It is important to increase dosage until symptoms are controlled or side effects become troublesome.

Mental and Emotional Disturbances of Everyday Practice—Depending on severity, *starting oral dosage* is 10 mg. t.i.d. or q.i.d., or 25 mg. b.i.d. or t.i.d. After a day or two, dosage may be increased by increments of 20 mg. to 50 mg. daily, at semiweekly intervals (increase should be more gradual in emaciated or senile patients) until achieving maximum clinical response. Continue dosage at this level for at least two weeks; then it can usually be reduced to a maintenance level. A daily dosage of 200 mg. is "average," but in some cases, such as discharged mental patients, daily dosages as high as 800 mg. may be necessary. *Starting intramuscular dose* is 25 mg. (1 cc.). If necessary, and if no hypotension occurs, repeat the initial dose in one hour. Subsequent dosages should be oral, starting at 25 mg. to 50 mg. t.i.d.

Alcoholism—***Severely agitated patients:*** *Starting intramuscular dose* is 25 mg. to 50 mg. (1-2 cc.). Repeat initial dose if necessary and if no hypotension occurs. Start subsequent oral dosages at 25 mg. to 50 mg. t.i.d. ***Agitated but manageable patients:*** *Starting oral dose* is 50 mg., followed by 25 mg. to 50 mg. t.i.d. For ambulatory patients with withdrawal symptoms or sober chronic alcoholics, *starting oral dosage* is 10 mg. t.i.d. or q.i.d., or 25 mg. b.i.d. or t.i.d. Patients in a stuporous condition should be allowed to sleep off some of the effects of the alcohol before 'Thorazine' is administered.

Hospitalized Psychiatric Patients—***Acutely agitated, manic, or disturbed patients:*** *Starting intramuscular dose* is 25 mg. (1 cc.). If no marked hypotension occurs, an additional 25 mg. to 50 mg. injection may be given after one hour. Subsequent intramuscular dosages may be increased gradually over a period of several days—even up to 400 mg. q4-6h in exceptionally severe cases—until the patient is controlled. (In elderly or emaciated patients the dosage should be increased more slowly than in other patients.) Usually the patient becomes quiet and cooperative within 24 to 48 hours after the initial dose, at which time oral doses may gradually be substituted for intramuscular doses (mg. for mg. or higher). Even if control is not complete, oral doses may gradually replace intramuscular doses. During this period, oral dosage should be increased rapidly until the patient is calm. Usually an *oral dose* of 500 mg. a day is sufficient but, if necessary, the dosage may be gradually increased still further to 2,000 mg. a day or higher. ***Less acutely agitated patients:*** *Starting oral dose* is 25 mg. t.i.d. Subsequently, increase the amount gradually until an effective dosage is reached—usually 400 mg. daily is sufficient. ***Duration of therapy:*** It is important to determine the optimal dosage regimen and to continue treatment long enough for maximum clinical response. Maximum improvement is sometimes not apparent until after weeks or even months of therapy.

PEDIATRIC DOSAGE AND ADMINISTRATION

Behavior Disorders—*Oral dosage* is on the basis of ¼ mg./lb. of body weight q4-6h, until symptoms are controlled (i.e., for 40 lb. child—10 mg. q4-6h). Calculate 'Thorazine' Syrup dosage at 10 mg./5 cc. tsp. *Rectal dosage* is on the basis of ½ mg./lb. of body weight q6-8h, p.r.n. (i.e., for 20-30 lb. child—half of a 25 mg. suppository q6-8h). *Intramuscular dosage* is on the basis of ¼ mg./lb. of body weight q6-8h, p.r.n. In children up to 5 years (or 50 lbs.)—not over 40 mg./day. In children 5-12 years (or 50-100 lbs.)—not over 75 mg./day. *Note:* In severe cases, 50-100 mg. daily has been used and, in older children, 200 mg. or more daily may be required.

IMPORTANT NOTES ON INJECTION

Except for acute ambulatory cases, parenteral administration should generally be reserved for bedfast patients. Parenteral administration should always be made with the patient lying down and remaining so for at least ½ hour afterward because of possible hypotensive effects. The injection should be given *slowly*, *deep* into the upper outer quadrant of the buttock. If irritation and pain at the site of injection are problems, dilution of 'Thorazine' Injection with physiologic saline solution or 2% procaine solution may be helpful. Subcutaneous administration is not advisable, and care should be taken to avoid injecting undiluted 'Thorazine' Injection into a vein. Intravenous administration is recommended only for severe hiccups and surgery. Because contact dermatitis has been reported, avoid getting the solution on hands or clothing.

SIDE EFFECTS

The drowsiness caused by 'Thorazine' may be unwanted in some patients. It is usually mild to moderate and disappears after the first or second week of therapy. If, however, drowsiness is troublesome, it can usually be controlled by lowering the dosage or by administering small amounts of dextro amphetamine.

Other side effects that have been reported occasionally are dryness of the mouth, nasal congestion, some constipation, miosis in a few patients and, very rarely, mydriasis. Mild fever (99°F.) may occur occasionally during the first days of therapy with large intramuscular doses. During 'Thorazine' therapy some patients have an increased appetite and gain weight. Usually these patients reach a plateau beyond which they do not gain further weight.

CAUTIONS

Jaundice: In the more than 14 million patients who have been treated with 'Thorazine' in the United States, the incidence of jaundice—regardless of indication, dosage, or mode of administration—has been low. Few cases have occurred in less than one week or after six weeks. Jaundice due to 'Thorazine' is of the so-called "obstructive" type; is without parenchymal damage; and is usually promptly reversible upon the withdrawal of 'Thorazine'. Because detailed liver function tests of 'Thorazine'-induced jaundice give a picture which mimics extrahepatic obstruction, exploratory laparotomy should be withheld until sufficient studies confirm extrahepatic obstruction.

Agranulocytosis: Agranulocytosis, although rare, has been reported in patients on 'Thorazine' therapy. Patients receiving 'Thorazine' should be observed regularly and asked to report at once the sudden appearance of sore throat or other signs of infection. If white blood counts and differential smears give an indication of cellular depression, the drug should be discontinued, and antibiotic and other suitable therapy should be instituted. Because most reported cases have occurred between the fourth and the tenth weeks of treatment, patients on prolonged therapy should be observed particularly during that period.

A moderate suppression of total white blood cells is sometimes observed in patients on 'Thorazine' therapy. If not accompanied by other symptoms, it is not an indication for discontinuing 'Thorazine'.

Potentiation: 'Thorazine' prolongs and intensifies the action of many central nervous system depressants, such as barbiturates and narcotics. Consequently, it is advisable to stop administration of such depressants before initiating 'Thorazine' therapy. Later the depressant agents may be reinstated, starting with low doses, and increasing according to response. Approximately ¼ to ½ the usual dosage of such agents is required when they are given in combination with 'Thorazine'. (However, 'Thorazine' does not potentiate the anticonvulsant action of barbiturates. In patients who are receiving anticonvulsants, the dosage of these agents—including barbiturates—should not be reduced if 'Thorazine' is started. Rather, 'Thorazine' should be started at a very low dosage and increased, if necessary.)

Hypotensive Effect: Postural hypotension and simple tachycardia may be noted in some patients. In these patients, momentary fainting and some dizziness are characteristic and usually occur shortly after the first parenteral dose, occasionally after a subsequent parenteral dose—very rarely after the first oral dose. In most cases, prompt recovery is spontaneous and all symptoms disappear within ½ to 2 hours with no subsequent ill effects. Occasionally, however, this hypotensive effect may be more severe and prolonged, producing a shock-like condition.

In consideration of possible hypotensive effects, the patient should be kept under observation (preferably lying down) for some time after the initial parenteral dose. If, on rare occasions, hypotension does occur, it can ordinarily be controlled by placing the patient in a recumbent position with head lowered and legs raised. If it is desirable to administer a vasoconstrictor, 'Levophed' and 'Neo-Synephrine'★ are the most suitable. Other pressor agents, including epinephrine, are not recommended because phenothiazine derivatives may reverse the usual elevating action of these agents and cause a further lowering of blood pressure.

Antiemetic Effect: The physician should always bear in mind that the antiemetic effect of 'Thorazine' may mask signs of overdosage of toxic drugs and may obscure diagnosis of conditions such as intestinal obstruction and brain tumor.

Dermatological Reactions: Dermatological reactions have been reported. Most have been of a mild urticarial type, suggesting allergic origin. Some of them appear to be due to photosensitivity, and it is advisable that patients on 'Thorazine' avoid undue exposure to the summer sun.

Neuromuscular Reactions: With very large doses of 'Thorazine', as frequently used in psychiatric cases over long periods, there have been a few patients who have exhibited neuromuscular reactions (extrapyramidal symptoms) which closely resemble parkinsonism. Such symptoms are reversible and usually disappear within a short time after the dosage has been decreased or the drug temporarily withdrawn. These neuromuscular reactions can also be controlled by the concomitant administration of standard antiparkinsonism agents.

Lactation: Moderate engorgement of the breast with lactation has been observed in female patients receiving very large doses of 'Thorazine'. This, however, is a transitory condition which disappears on reduction of dosage or withdrawal of the drug.

CONTRAINDICATIONS

In comatose states due to central nervous system depressants (alcohol, barbiturates, narcotics, etc.), and also in patients under the influence of large amounts of barbiturates or narcotics.

AVAILABLE

Tablets, 10 mg., 25 mg., 50 mg. and 100 mg., in bottles of 50, 500 and 5000; 200 mg., for use in mental hospitals, in bottles of 500 and 5000. (Each tablet contains chlorpromazine hydrochloride, 10 mg., 25 mg., 50 mg., 100 mg., or 200 mg.)

Ampuls, 1 cc. and 2 cc. (25 mg./cc.), in boxes of 6, 100 and 500. (Each cc. contains, in aqueous solution, chlorpromazine hydrochloride, 25 mg.; ascorbic acid, 2 mg.; sodium bisulfite, 1 mg.; sodium sulfite, 1 mg.; sodium chloride, 6 mg.)

Multiple-dose Vials, 10 cc. (25 mg./cc.), in boxes of 1, 20 and 100. (Each cc. contains, in aqueous solution, chlorpromazine hydrochloride, 25 mg.; ascorbic acid, 2 mg.; sodium bisulfite, 1 mg.; sodium sulfite, 1 mg.; sodium chloride, 1 mg. Contains benzyl alcohol, 2%, as preservative.)

Spansule® capsules, 30 mg., 75 mg., 150 mg. and 200 mg., in bottles of 30, 250 and 1500; also 300 mg., in bottles of 30 and 1500. (Each 'Spansule' capsule contains chlorpromazine hydrochloride, 30 mg., 75 mg., 150 mg., 200 mg., or 300 mg.)

Syrup, 10 mg./teaspoonful (5 cc.), in 4 fl. oz. bottles. (Each 5 cc. contains chlorpromazine hydrochloride, 10 mg.)

Suppositories, 25 mg. and 100 mg., in boxes of 6. (Each suppository contains chlorpromazine, 25 mg. or 100 mg.; glycerin, glyceryl monopalmitate, glyceryl monostearate, hydrogenated cocoanut oil fatty acids, hydrogenated palm kernel oil fatty acids, lecithin.)

Concentrate (for hospital use), 30 mg./cc., in 4 fl. oz. bottles, packages of 12 and 36; and in 1 gal. bottles. (Each cc. contains chlorpromazine hydrochloride, 30 mg.)

★'Levophed' and 'Neo-Synephrine' are the trademarks (Reg. U.S. Pat. Off.) of Winthrop Laboratories for its brands of levarterenol and phenylephrine respectively.

FOR QUICK CONTROL OF AGITATED BEHAVIOR...

With 'Thorazine'—particularly the injectable form—you can quickly control severe agitation in patients such as alcoholics, seniles and hyperkinetic children. Belligerence and excitement usually give way promptly to more rational behavior, and the patient becomes receptive to guidance and counselling.

'Thorazine' is so effective in calming agitated patients because it provides:

- a potent tranquilizing action, which controls emotional agitation
- an initial sedative effect for control of physical hyperactivity during the first few days of therapy

'Thorazine' has been used in over 14 million patients in the United States during the past 7 years and, despite the advent of many newer agents, continues in widespread use in both office and hospital practice. This experience confirms that, in the vast majority of patients, the potential benefits of 'Thorazine' far outweigh its possible hazards.

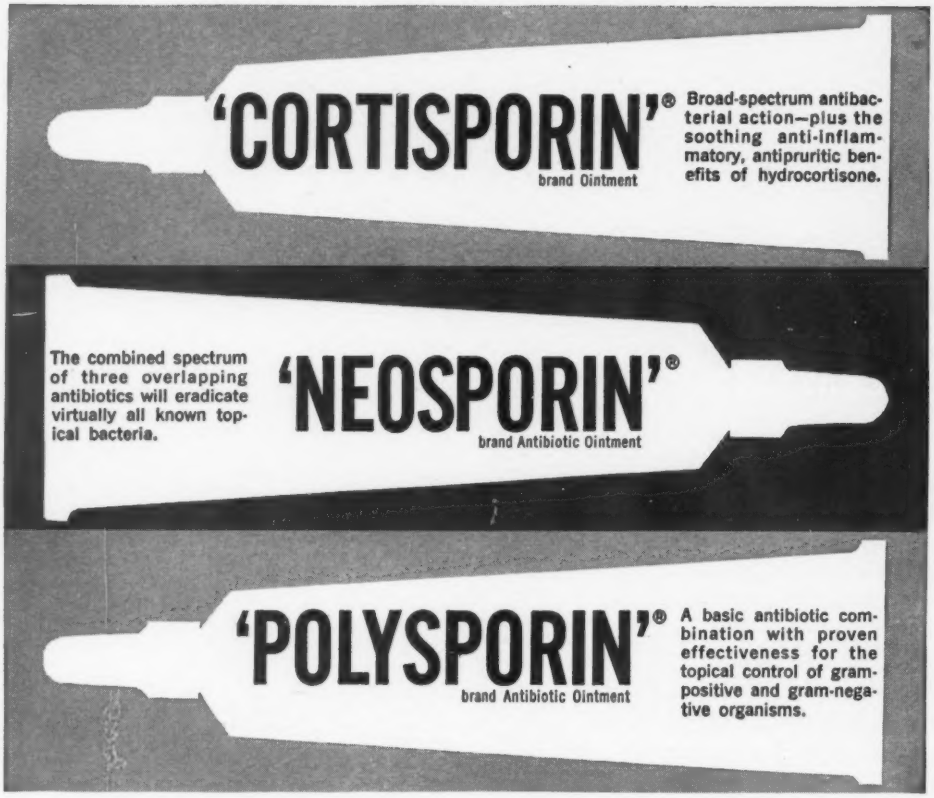
THORAZINE®

brand of chlorpromazine

a fundamental drug in medicine

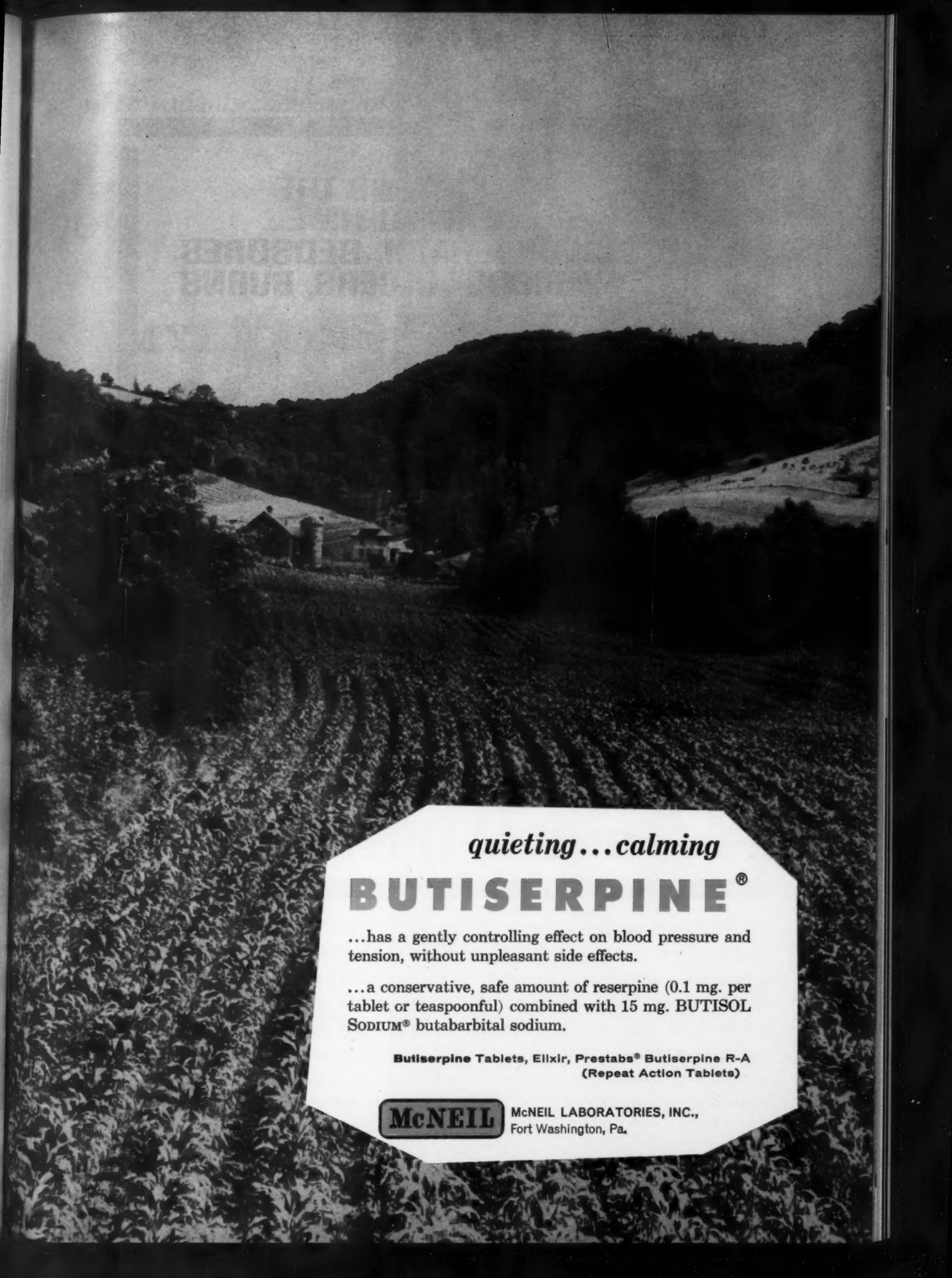
SK&F Smith Kline & French Laboratories

'B. W. & Co.' 'Sporin' Ointments rarely sensitize . . . give decisive bactericidal action for most every topical indication


'CORTISPORIN'®
brand Ointment
Broad-spectrum antibacterial action—plus the soothing anti-inflammatory, antipruritic benefits of hydrocortisone.
The combined spectrum of three overlapping antibiotics will eradicate virtually all known topical bacteria.
'NEOSPORIN'®
brand Antibiotic Ointment
'POLYSPORIN'®
brand Antibiotic Ointment
A basic antibiotic combination with proven effectiveness for the topical control of gram-positive and gram-negative organisms.

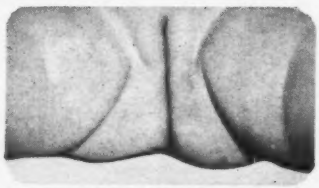
Contents per Gm.	'Polysporin'®	'Neosporin'®	'Cortisporin'®
'Aerosporin'® brand Polymyxin B Sulfate	10,000 Units	5,000 Units	5,000 Units
Zinc Bacitracin	500 Units	400 Units	400 Units
Neomycin Sulfate	—	5 mg.	5 mg.
Hydrocortisone	—	—	10 mg.
Supplied:	Tubes of 1 oz., ½ oz. and ⅛ oz. (with ophthalmic tip)	Tubes of 1 oz., ½ oz. and ⅛ oz. (with ophthalmic tip)	Tubes of ½ oz. and ⅛ oz. (with ophthalmic tip)

BURROUGHS WELLCOME & CO. (U.S.A.) INC., Tuckahoe, New York


quieting...calming
BUTISERPINE®
...has a gently controlling effect on blood pressure and tension, without unpleasant side effects.
...a conservative, safe amount of reserpine (0.1 mg. per tablet or teaspoonful) combined with 15 mg. BUTISOL SODIUM® butabarbital sodium.
Butiserpine Tablets, Elixir, Prestabs® Butiserpine R-A (Repeat Action Tablets)
McNEIL
McNEIL LABORATORIES, INC.,
Fort Washington, Pa.

SHORTENS THE HEALING TIME... DIAPER RASH, BEDSORES VARICOSE ULCERS, BURNS

Before application of A and D Ointment—Typical diaper rash with excoriation of skin.



After application of A and D Ointment—Diaper rash has completely disappeared within one week.

Before application of A and D Ointment—Treatment-resistant varicose ulcer in elderly obese patient.

After five weeks of daily treatment with A and D Ointment—Ulcer completely healed.

Before application of A and D Ointment—Second and third degree burns caused by flaming gasoline.

After pressure gauze dressings of A and D Ointment, changed weekly—Completely healed, minimal scar tissue and no contractures.

A and D OINTMENT

REG. T.M.

completely safe, highly effective in soothing and healing a wide range of skin disorders

A and D Ointment serves a useful purpose in every practice. It promotes granulation and epithelization in ulcers, burns, bedsores and wounds. In diaper rash, it instantly provides soothing relief and helps to quicken healing. / A and D Ointment is eminently safe—may be applied liberally to even the most delicate tissues. It will not stain the skin or wash away in body secretions. Easily laundered from clothing. / Available in 1½ or 4 oz. tubes; 1 or 5 lb. jars. Also available: A and D Ointment with Prednisolone, in 10 and 25 gm. tubes.

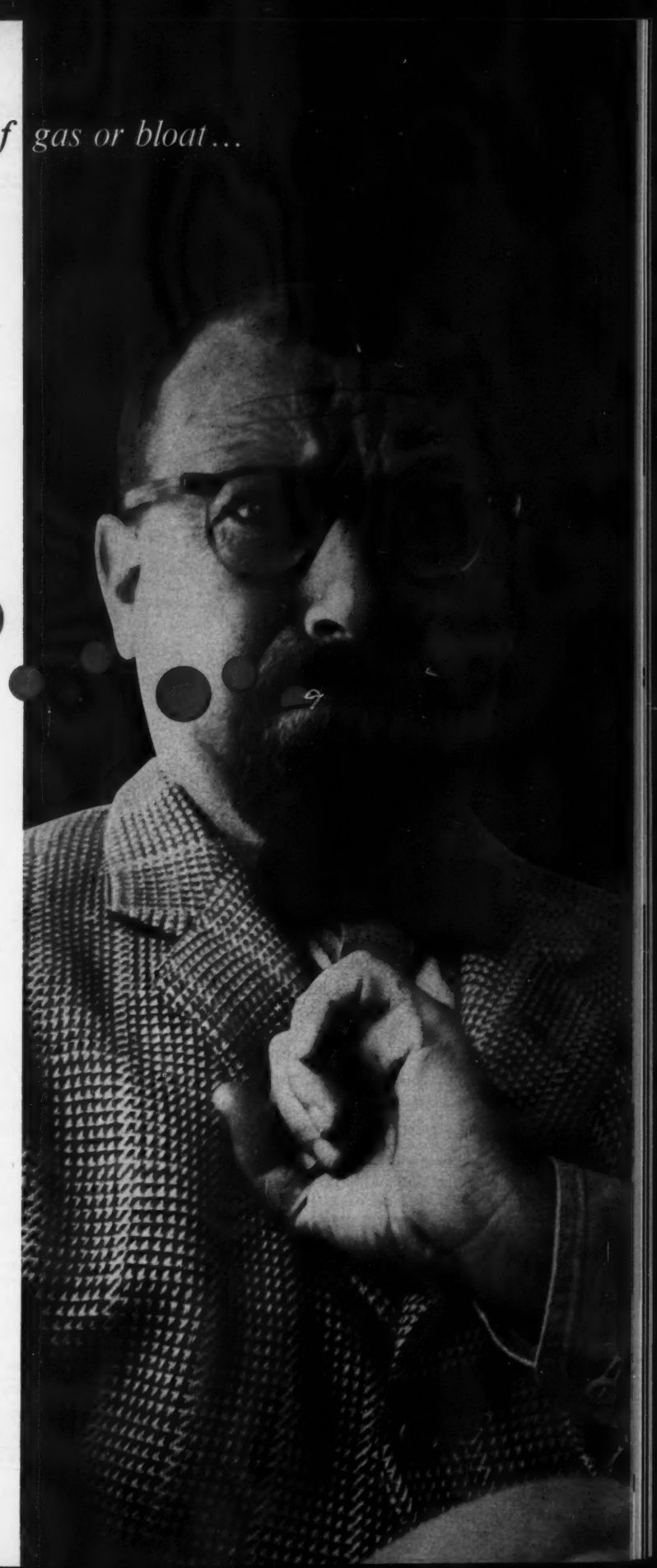
WHITE LABORATORIES, INC. / KENILWORTH, NEW JERSEY

White

when patients complain of gas or bloat...

new

MYLICON®



MYLICON relieves the gastrointestinal distress produced by entrapped gas. MYLICON's defoaming action changes the surface tension of gas bubbles, permitting them to coalesce. This gas is released and eliminated by belching or passing flatus. MYLICON is physiologically inert, nontoxic.

Gas entrapment and distention can result from Spastic Colitis, Aerophagia, Postoperative Gas, Postgastrectomy Syndrome, Hyperacidity, Hiatus Hernia, Diverticulitis, Gastric and Duodenal Ulcers.

Pleasant tasting, soft chewable tablets can be taken without water.

One white scored tablet contains:
Methylpolysiloxane, a silicone.....40 mg.

DOSAGE: one tablet after each meal and at bedtime.

SUPPLIED: bottles of 100 and 500 tablets at all pharmacies.

References: "Intestinal Gas and Bloating; Treatment with Methylpolysiloxane," Am. Pract. & Dig. of Treat., 11:52. (Jan.) 1960.

"Use of Silicone in the Treatment of Intestinal Gas and Bloating," J.A.M.A., 174:2052, (Dec. 17) 1960.

also *NEW* **MYLICON® DROPS**
for infant colic caused by excessive air swallowing or inability to belch or pass flatus.

MYLICON DROPS (0.3 cc. to 0.6 cc.) can be given directly from the dropper or added to each feeding.

Each 0.6 cc. represents 40 mg. of methylpolysiloxane, equivalent to one MYLICON tablet.

Available at all pharmacies in bottles of 30 cc. of drops.

Stuart

THE STUART COMPANY
PASADENA, CALIFORNIA

11881/4100

in severe drug and food sensitivity... rapid relief and control of symptoms on short-term therapy with Decadron®

Brief treatment with DECADRON—orally or parenterally—can provide rapid and effective control of allergic emergencies and acute allergic disorders such as reactions to foods, drugs, plants, weeds, and animals. In 40 patients given Injection DECADRON Phosphate, "subjective improvement was often noticed within one hour and objective improvement recorded within four hours."[1] Therapeutic doses of steroids may help prevent recurrences of severe allergic states, without interfering with desensitization or other immunity procedures.[2]

Before prescribing or administering DECADRON, the physician should consult the detailed information on use accompanying the package or available on request.

References: 1. Grater, W. C.: Southern M. J. 53:1144, 1960. 2. Feinberg, S. M.: Med. Sci. 6:(No. 3)181, 1959.

Supplied: As 0.75 mg. and 0.5 mg. scored, pentagon-shaped tablets in bottles of 100 and 1000. As Injection DECADRON Phosphate in 5 cc. vials, each cc. containing 4 mg. of dexamethasone 21-phosphate as the disodium salt; inactive ingredients: 8 mg. creatinine, 10 mg. sodium citrate; sodium hydroxide to pH 7.8, and water for injection q.s. 1 cc.; preservatives: 0.32 per cent sodium bisulfite and 0.5 per cent phenol. DECADRON is a trademark of Merck & Co., Inc.

MERCK SHARP & DOHME
Division of Merck & Co., Inc., West Point, Pa.

Decadron®
TREATS MORE PATIENTS MORE EFFECTIVELY

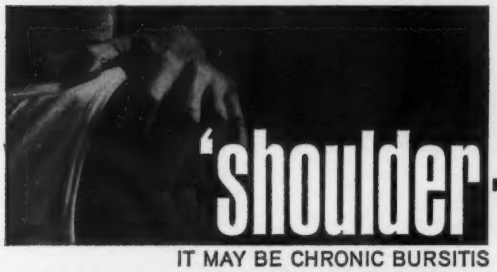
DECADRON: Recommended dosage schedule in the treatment of drug and food sensitivity reactions		
time	**amount**	**administration**
1st day	one to two cc. (4 to 8 mg.) Injection DECADRON Phosphate intramuscular	repeated as necessary (in substituting tablet therapy, give the first oral dose four or five hours before the final parenteral dose.)
2nd day	two 0.75 mg. Tablets DECADRON	b.i.d.
3rd day	two 0.75 mg. Tablets DECADRON	b.i.d.
4th day	one 0.75 mg. Tablet DECADRON	b.i.d.
5th day	one 0.75 mg. Tablet DECADRON	per day
6th day	one 0.75 mg. Tablet DECADRON	per day
7th day	RETURN VISIT	

in any rheumatic 'itis'

Schering

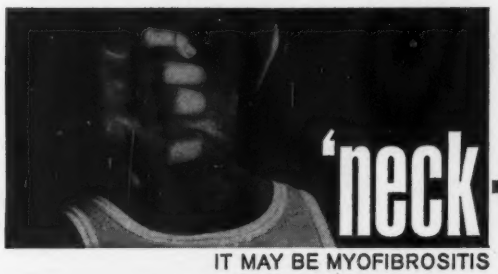
'hand-itis'

IT MAY BE EARLY RHEUMATOID ARTHRITIS



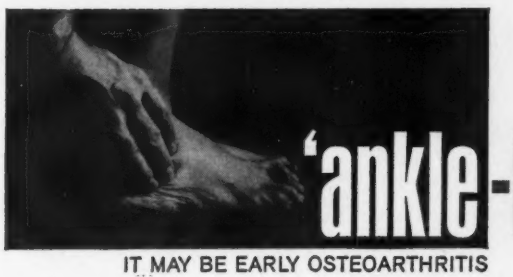
'shoulder-itis'

IT MAY BE CHRONIC BURSITIS



'neck-itis'

IT MAY BE MYOFIBROSITIS



'ankle-itis'

IT MAY BE EARLY OSTEOARTHRITIS

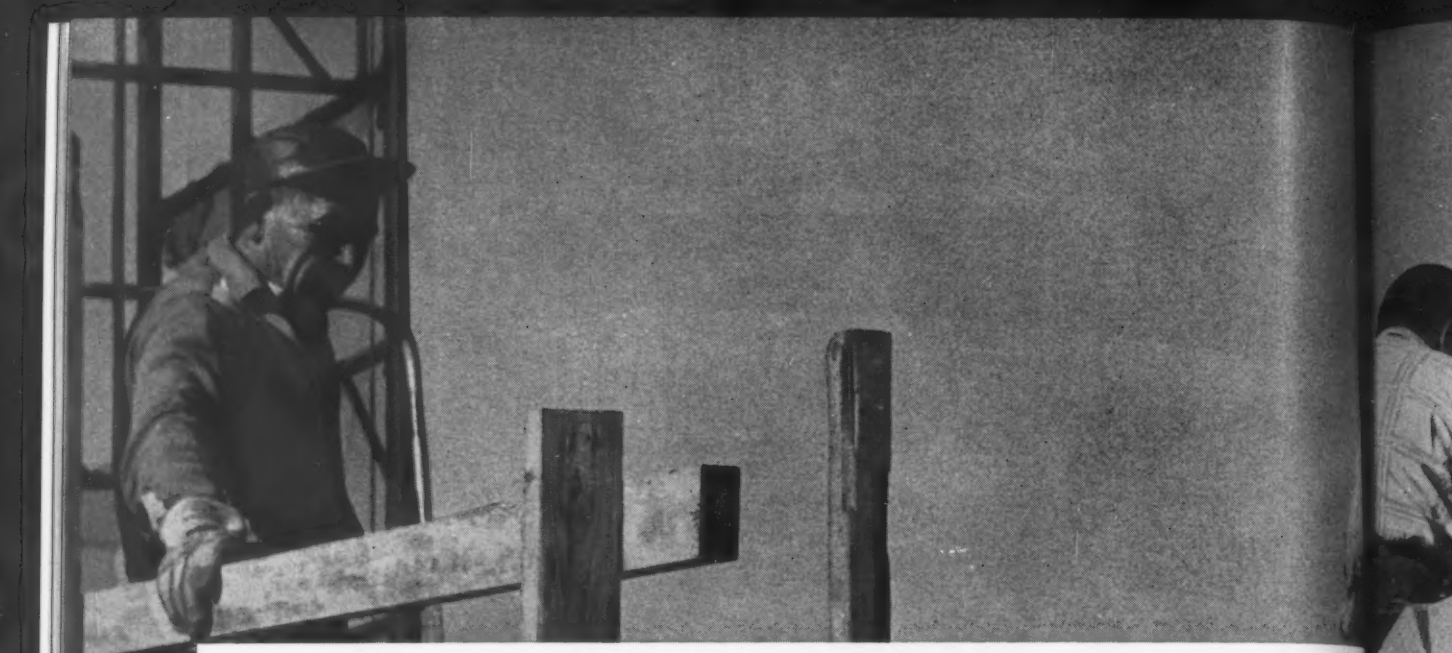
The favored corticoid-salicylate compound. For more effective and comprehensive, yet conservative, treatment than either steroids or salicylates alone... the outstanding anti-inflammatory effect of prednisone[1]...the supportive antirheumatic action of aspirin[2,3]...to bring rapid pain relief and quiet the inflammatory process. SIGMAGEN offers less likelihood of treatment-terminating side effects.[2] SIGMAGEN is available in bottles of 100 and 1000.

METICORTEN® (prednisone)	safer, reduced dosage	0.75 mg.
Acetylsalicylic acid	supportive anti-inflammatory-analgesic	325 mg.
Aluminum hydroxide	a buffer for better toleration	75 mg.
Ascorbic acid	anti-stress supplementation	20 mg.

References: 1. Cohen, A., et al.: J.A.M.A. 165:225, 1957. **2.** Spies, T. D., et al.: J.A.M.A. 159:645, 1955. **3.** Stecher, R. M.: Panel Discussion, Ohio M. J. 52:1037, 1956.

Remission-in any rheumatic 'itis' Sigmagen®

H-389



Put your low-back patient back on the payroll

Soma's prompt relief of pain and stiffness can get your low-back patients back to work in days instead of weeks

Soma is unique because it combines the properties of an effective muscle relaxant and an independent analgesic in *a single drug.* Unlike most other muscle relaxants, which can only relax muscle tension, Soma attacks both phases of the pain-spasm cycle at the same time.

Thus with Soma, you can break up both pain and spasm fast, effectively . . . help give your patient the two things he wants most: relief from pain and rapid return to full activity.

Soma is notably safe. Side effects are rare. Drowsiness may occur, but usually only with higher dosages. Soma is available in 350 mg. tablets. USUAL DOSAGE: 1 TABLET Q.I.D.

The muscle relaxant with an independent pain-relieving action

SOMA®

(carisoprodol, Wallace)

Wallace Laboratories, Cranbury, New Jersey

How you can help save your patients a month's pay

Kestler reports in J.A.M.A. (April 30, 1960) that conventionally treated low-back syndrome patients required an average of 41 days for full recovery (range: 3 to 90 days). The addition of Soma therapy in this comparative investigation reduced the average to 11.5 days (range: 2 to 21 days). With Soma, patients averaged full recovery 30 days sooner.

CONCLUSIONS
of another clinical study[1] with
QUINAGLUTE®
DURA-TAB S. M.®
the only oral Sustained Medication* Quinidine Gluconate 5 gr. (0.33 Gm.)
IN ATRIAL FIBRILLATION
• effectively maintained normal sinus rhythm in almost all cases.
• q. 12 h. dosage usually sufficed to maintain adequate serum levels.
• no night dosage needed — a single late evening dose avoided recurrence of fibrillation.
• from 8 to 12 hours after administration, "definitely higher" serum quinidine levels than with ordinary quinidine sulfate.
QUINAGLUTE DURA-TAB S.M. — a quinidine of choice in atrial fibrillation, flutter, premature contractions, auricular tachycardia. Bottles of 30, 100 and 250.
For sample supply and reprint write...
WYNN PHARMACAL CORPORATION
Lancaster Ave. at 51st St., Phila. 31, Pa
For dosage, etc.
PDR
Page 821
*U. S. Patent 2,895,881
1. Greif, E. and Scheuer, J.: J. Mount Sinai Hosp., Nov./Dec. 1960

"wearability"

355 cc. (12 Ounces)
List No. 344
MAALOX®
RORER
(MAGNESIUM-ALUMINUM HYDROXIDE GEL)
ANTACID — DEMULCENT
NON-CONSTIPATING
A colloidal suspension of Magnesium and Aluminum Hydroxides useful for the relief of gastric hyperacidity.
Shake Well Before Using
Average adult dose: As antacid and protective, two to four teaspoonfuls may be given in water or milk twenty minutes after meals and at bedtime. Use as directed by your physician. Maalox should not be used in patients who are severely debilitated or suffering from kidney failure.
KEEP BOTTLE TIGHTLY CLOSED
KEEP FROM FREEZING
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WILLIAM H. RORER, Inc.
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NO TASTE FATIGUE
EXCELLENT RESULTS
NO CONSTIPATION

the most widely prescribed and most wearable of all antacids

suspension tablets

Tablet Maalox No. 1 equivalent to 1 teaspoon Suspension
Tablet Maalox No. 2 equivalent to 2 teaspoons Suspension

✓ from depression to **the right frame of mind**

✓ continuous, 24-hour cerebral oxygenation for the aging patient. By stimulating respiratory and circulatory function, GERONIAZOL TT* relieves mental confusion, depression, anxiety, and emotional instability—frequent problems in patients after forty—due to presenile changes in the vasculature of the brain. Notable benefit usually is seen within one to three weeks of therapy. It improves appetite, sleep pattern, and outlook—and GERONIAZOL TT* is non-hypertensive, non-excitatory.

Neither a tranquilizer nor a psychic energizer, GERONIAZOL TT* provides a physiologic stimulation of the cerebrum to permit the patient to adjust to his surroundings, become part of life itself again—and *attain the right frame of mind.*

References: 1. Curran, T. R., and Phelps, D. K.: Am. Pract. & Dig. Treat. *11:* 617, 1960. 2. Levy, S.: J.A.M.A. *153:* 1260, 1953. 3. Connolly, R.: W. Va. Med. J. *56:* 263, 1960.

Each TEMPOTROL contains: Pentylenetetrazol, 300 mg.; and Nicotinic Acid, 150 mg.

Indications: Respiratory and circulatory stimulant for the aged and debilitated with symptoms of mental confusion, depression, anxiety or arteriosclerotic psychosis.

Contraindications: None known in recommended dosage.

Dosage: One GERONIAZOL TT* tablet, b. i. d.

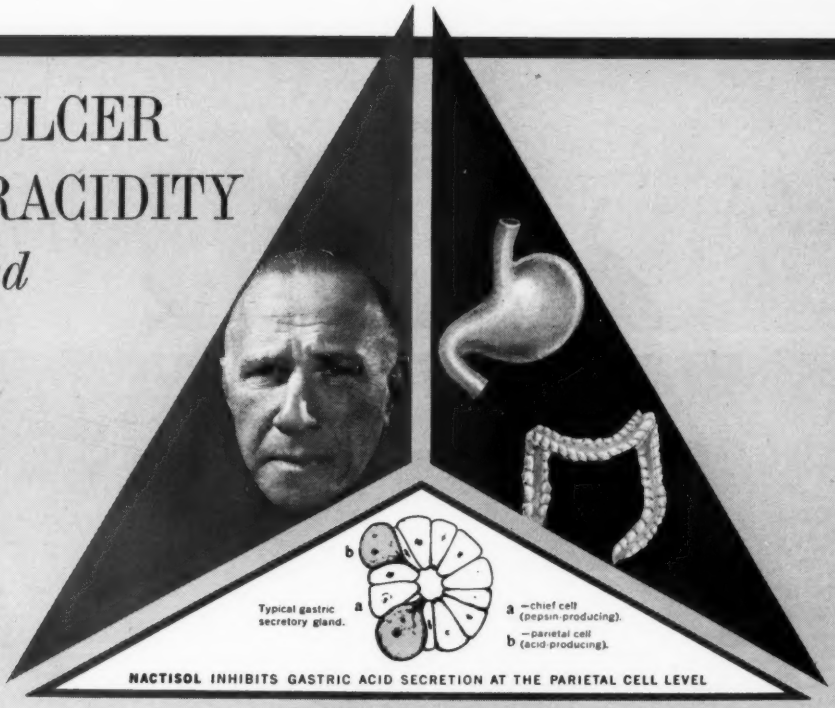
Supplied: Bottles of 42 tablets (3 weeks' treatment).

GERONIAZOL® TT*

*TEMPOTROL® (Time Controlled Therapy)

COLUMBUS PHARMACAL COMPANY affiliate of PHILIPS ROXANE, INC.
Columbus 16, Ohio

IN PEPTIC ULCER AND HYPERACIDITY *with associated* tension and nervousness


b
Typical gastric secretory gland.
a
a —chief cell (pepsin-producing).
b —parietal cell (acid-producing).
NACTISOL INHIBITS GASTRIC ACID SECRETION AT THE PARIETAL CELL LEVEL

NACTISOL*

- suppresses gastric acid secretion at the parietal cell level
- decreases gastrointestinal hypermotility
- relieves nervousness and tension

NACTISOL combines:

NACTON® 4 mg. (poldine methylsulfate†) — new inhibitor of gastric acid secretion and hypermotility "...reduces the total output of gastric HCl by about 60%"[1]

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smooth, predictable sedation of *6 hours' duration*

- Side effects with NACTISOL therapy have been minimal.[3-5]

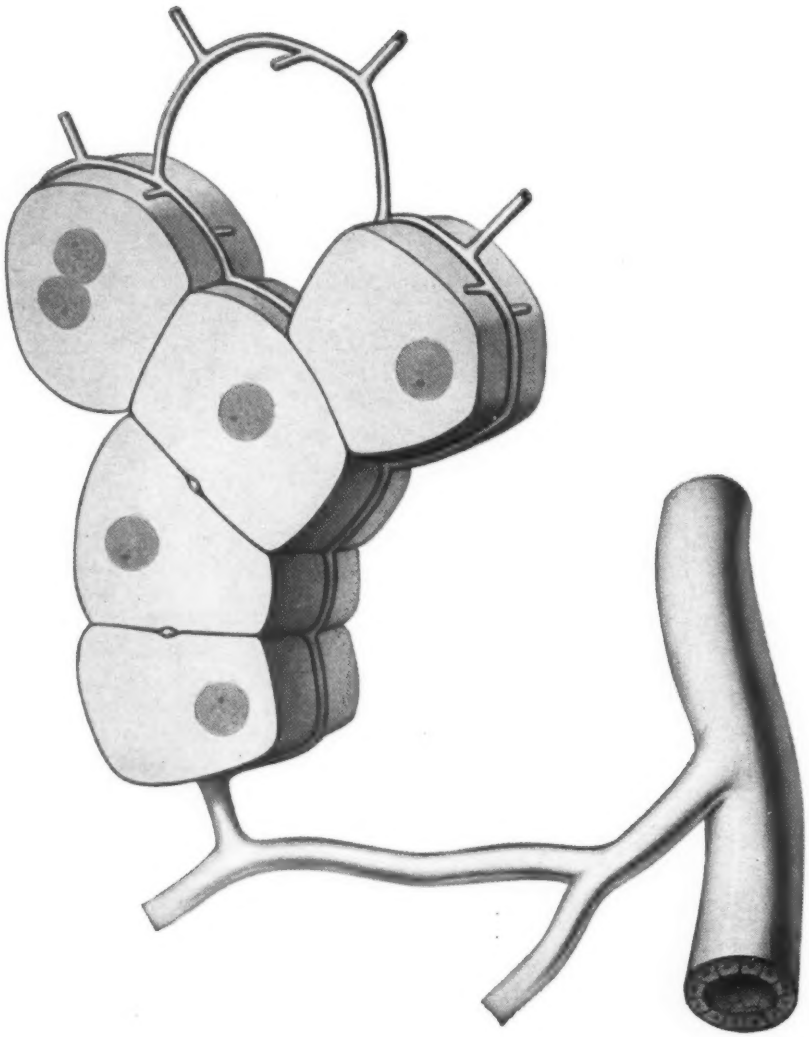
NACTISOL*...in scored, yellow tablets

References

1. Douthwaite, A. H.: The Development of the Treatment of Duodenal Ulcer, Proc. Roy. Soc. Med. *51*:1063-1068 (December) 1958. 2. Batterman, R. C., Grossman, A. J., Leifer, P., and Mouratoff, G. J.: Clinical Re-evaluation of Daytime Sedatives, Postgrad. Med. *26*:502-509 (October) 1959. 3. Steigmann, F.: Clinical Report to McNeil Laboratories. 4. Lorber, S. H.: Clinical Report to McNeil Laboratories, December 6, 1960. 5. Rider, J. A.: Clinical Report to McNeil Laboratories.

McNEIL McNEIL LABORATORIES, INC., Fort Washington, Pa.

*Trademark
†U. S. Patent



IMPROVE HEPATO-BILIARY FUNCTION AT THE CELLULAR LEVEL WITH CHOLAN-HMB®

Cholan-HMB contains chemically pure oxidized bile acid to stimulate the production of a large volume of thin bile, flush the biliary tree, improve digestion of fats, and relieve the symptoms of dyspepsia, constipation and indigestion caused by intrahepatic biliary stasis.

To counteract spasm of the biliary sphincters and relax the gallbladder, Cholan-HMB contains spasmolytic homatropine methylbromide. The mild sedative component helps to calm the tense, emotional patient with hepatobiliary dysfunction. Cholan-HMB provides physiologic and symptomatic relief of biliary stasis in primary biliary dyskinesia, pre- and postcholecystectomy syndrome, cholangitis, and digestive disturbances of pregnancy.

Cholan-HMB® — dehydrocholic acid, Maltbie, 250 mg.; 2.5 mg. homatropine methylbromide, and 8 mg. phenobarbital. 1 or 2 tablets t.i.d. after meals.

Cholan-V® — dehydrocholic acid, Maltbie, 250 mg., and 5 mg. homatropine methylbromide. 1 or 2 tablets t.i.d. after meals.

Cholan-DH® — dehydrocholic acid, Maltbie, 250 mg. 1 or 2 tablets t.i.d. after meals.

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Maltbie

Maltbie Laboratories Division, Wallace & Tiernan Inc., Belleville 9, New Jersey

PCN-11

ANNOUNCING... A POTENT
ANTIDEPRESSANT
WITH EFFECTIVE
ANTI-ANXIETY
PROPERTIES...

NEW

ELAVIL®

AMITRIPTYLINE HYDROCHLORIDE

MSD

new...a potent antidepressant with effective anti-anxiety properties

ELAVIL

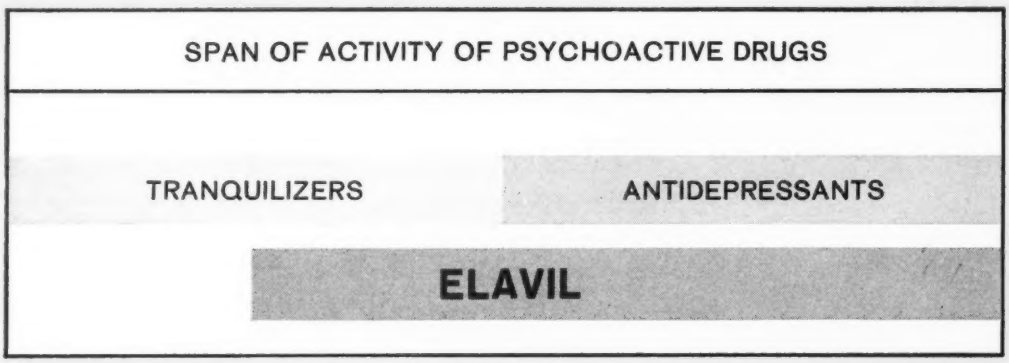
AMITRIPTYLINE HYDROCHLORIDE

RELATIVE UTILITY IN MANAGEMENT OF DEPRESSED PATIENTS

	TARGET SYMPTOMS OF DEPRESSION:			
Class of compounds	*Anxiety*	*Insomnia*	*Depression*	*Over-all relief of symptoms*
TRANQUILIZERS	"Failure of the tranquilizers to produce satisfactory results is due in many cases to their being prescribed for depression, especially depression masked by the more prominent symptoms of anxiety. The underlying depression may be deepened."[1]			+ / −
ANTIDEPRESSANTS			"CNS stimulants and anti-depressants, if given to anxious patients, will increase the anxiety...."[1]	+
ELAVIL	"...this drug [ELAVIL] acted both as a tranquilizer and as an anti-depressant..."[2] Many physicians customarily treat anxious or depressed patients with a combination of an antidepressant and a tranquilizer. This is seldom necessary when prescribing ELAVIL because it has both antidepressant and anti-anxiety properties.			++

AVIL®

effective in patients with depression...
particularly useful in those with predominant symptoms of anxiety and tension...provides prompt relief of anxiety and insomnia associated with depression



INDICATIONS: manic-depressive reaction – depressed phase; involutional melancholia; reactive depression; schizo-affective depressions; neurotic depressive reaction; and these target symptoms: anxiety; depressed mood; insomnia; psychomotor retardation; functional somatic complaints; loss of interest; feelings of guilt; anorexia. May be used whether the emotional difficulty is a manifestation of neurosis or psychosis,[4] and in ambulatory or hospitalized patients.[3, 4, 5]

USUAL ADULT ORAL DOSAGE: Initial, 25 mg. three times a day, until a satisfactory response is noted. Many patients improve rapidly, although some depressed patients may require four to six weeks of therapy before obtaining maximum benefit. In severely depressed patients, as much as 150 mg. per day may be given. **Maintenance,** 25 mg. two to four times a day. Some patients may be maintained on 10 mg. four times a day. The natural course of depression is often many months in duration. Accordingly, it is appropriate to continue maintenance therapy for at least three months after the patient has achieved satisfactory improvement in order to lessen the possibility of relapse, which may occur if the patient's depressive cycle is not complete. In the event of relapse, therapy with ELAVIL may be reinstituted.

ELAVIL is not a monoamine oxidase (MAO) inhibitor. No evidence of drug-induced jaundice or agranulocytosis has been noted. Side effects (drowsiness, dizziness, nausea, excitement, hypotension, fine tremor, jitteriness, headache, heartburn, anorexia, increased perspiration, and skin rash), when they occur, are usually mild. However, as with all new therapeutic agents, careful observation of patients is recommended. As with other drugs possessing significant anticholinergic activity, ELAVIL is contraindicated in patients with glaucoma.

SUPPLY: Tablets, 10 mg. and 25 mg., in bottles of 100. **Injection** (intramuscular), 10 mg. per cc., 10-cc. vials.

REFERENCES: 1. Perloff, M. M., and Levick, L. J.: Clinical Med. 7:2237, Nov. 1960. 2. Freed, H.: Am. J. Psychiat. 117:455, Nov. 1960. 3. Dorfman, W.: Psychosomatics 1:153, May-June 1960. 4. Ayd, F. J., Jr.: Psychosomatics 1:320, Nov.-Dec. 1960. 5. Barsa, J. A., and Saunders, J. C.: Am. J. Psychiat. 117:739, Feb. 1961.

Before prescribing or administering ELAVIL, the physician should consult the detailed information on use accompanying the package or available on request.

MSD MERCK SHARP & DOHME, DIVISION OF MERCK & CO., INC., WEST POINT, PA.

ELAVIL IS A TRADEMARK OF MERCK & CO., INC.

NEW comprehensive digestant with the *most potent* enzyme available for digestion of

—also unsurpassed potency for digestion of starch, protein and cellulose

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—the *only* digestant with fat-splitting lipase activity 12 times as great as that of Pancreatin N.F.

When the question is digestion because of your patient's inability to handle fat, starch, protein or cellulose, you can provide dependable relief with COTAZYM-B, which contains the essential pancreatic enzymes lipase, trypsin and amylase, plus bile salts and cellulase. A daily dose of 6 COTAZYM-B tablets is sufficient to emulsify and digest 50 Gm. of dietary fat, and to digest all of the protein and starch in a typical diet (100 Gm. protein, 250 Gm. starch) and 480 mg. cellulose.

Dosage: **1 or 2 tablets with water just before each meal.**

Supply: **Bottles of 48 tablets.**

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NEW Cotazym®-B

Lipancreatin Bile Salts Cellulase

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A product of original Organon research, lipancreatin provides *for the first time in digestant preparations a known, constant amount of fat-digesting lipase* in addition to trypsin and amylase. It surpasses in assayable digestive activity *all* presently available pancreatin preparations.

In over six years of clinical use...

Proven
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Effective
for relief of anxiety and tension

Outstandingly Safe

1. simple dosage schedule relieves anxiety dependably – without the unknown dangers of "new and different" drugs
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3. no cumulative effects in long-term therapy
4. does not produce depression, Parkinson-like symptoms, jaundice or agranulocytosis
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meprobamate (Wallace)

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stop pain with Nupercainal®

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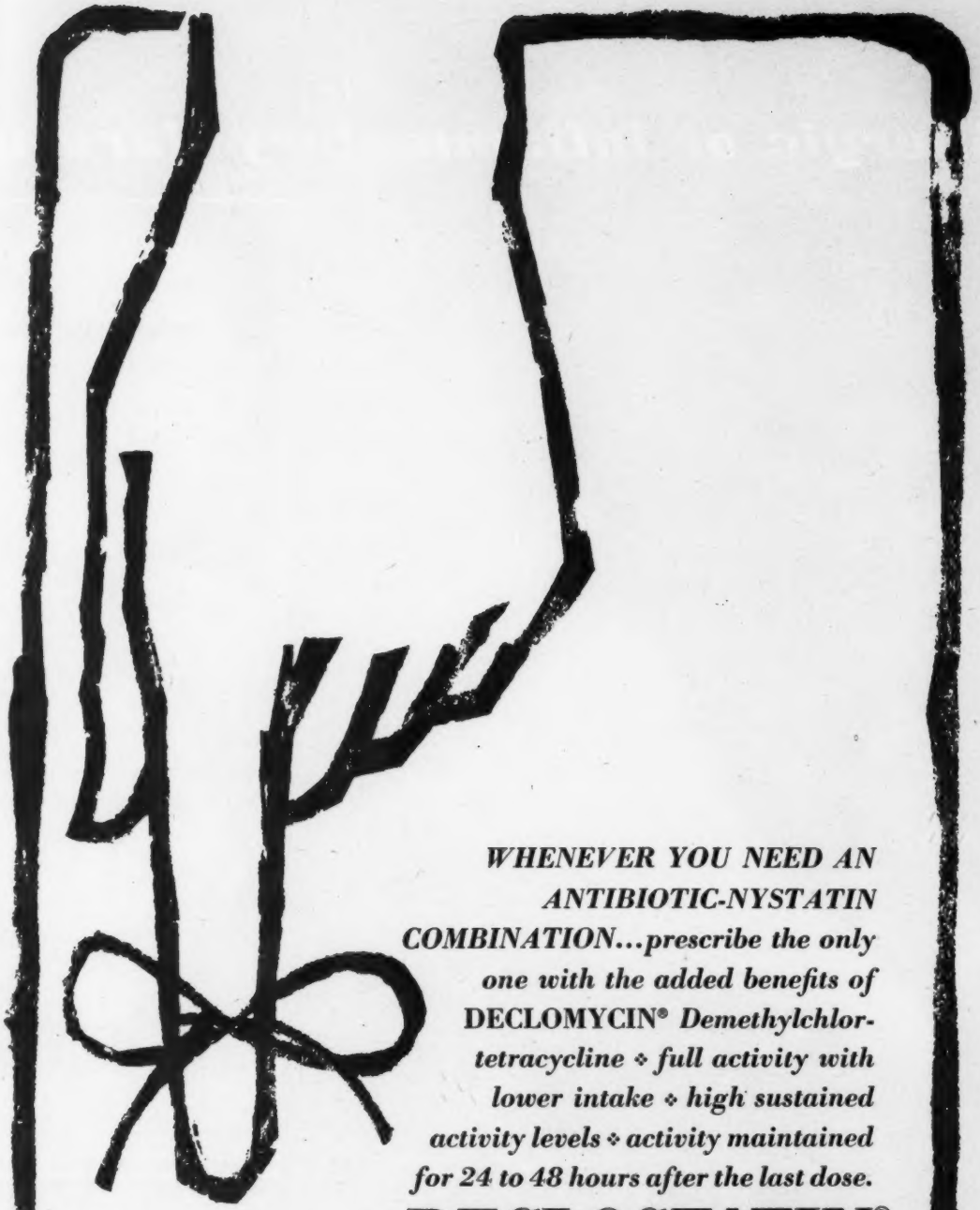
NUPERCAINAL SUPPOSITORIES
exact dosage • fast acting



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***remember* DECLOSTATIN®**

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Request complete information on indications, dosage, precautions and contraindications from your Lederle representative, or write to Medical Advisory Department.

LEDERLE LABORATORIES, A Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

Allergic or inflammatory flare-up!

Female, 41. Dx: dermatitis venenata. Contactant: calamine-antihistamine lotion applied for contact dermatitis—Rx Celestone Tablets, 0.6 mg. Photograph prior to Rx.

Step-down dosage of 1 tab. q.i.d. for 2 days, 1 tab. t.i.d. for 2 days, 1 tab. b.i.d. for 2 days and 1 tab. daily for 8 days. Results: condition completely cleared. Side Effects: none. Photograph after 72 hours of Celestone therapy. (Photographs courtesy of M. M. Nierman, M.D., Calumet City, Ill.)

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Rapid remission with new Celestone

the first major advance in corticosteroid therapy in over 2½ years

Clinical worth: CELESTONE provides greatly enhanced antiallergic and anti-inflammatory effects with significantly lower mg. dosages. Its efficacy and safety have been established by 20 months of pre-introductory clinical trials in such steroid-responsive disorders as:

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- weight loss
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- vertigo
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- potassium excretion

Improved response: CELESTONE also offers the advantage of providing an opportunity to restore "lost" or diminished control in patients receiving other steroids.

For complete details, consult latest Schering literature available from your Schering Representative or the Medical Services Department, Schering Corporation, Bloomfield, New Jersey.

Bibliography: 1. Goldman, L.: Investigation of a New Steroid in Dermatology. Paper presented at First Symposium on the Clinical Application of Betamethasone: A New Corticosteroid, New York City, May 8, 1961. 2. Nierman, M. M.: The Use of Betamethasone in Dermatology. *Ibid.* 3. Gant, J. Q., and Gould, A. H.: Betamethasone: A Clinical Study. *Ibid.* 4. Frank, L.: The Place of Betamethasone in Dermatologic Practice. *Ibid.* 5. Hampton, S. F.: Betamethasone—A New Steroid in Allergy: A Preliminary Report. *Ibid.* 6. Bukantz, S. C.: Observations on the Use of Betamethasone in the Intractable Asthmatic Child. *Ibid.* 7. Bedell, H.: A New Systemic Steroid in the Treatment of Allergies in Office Practice. *Ibid.* 8. Schwartz, E.: Clinical Evaluation of Betamethasone in Chronic Intractable Bronchial Asthma. *Ibid.* 9. Kammerer, W. H.: Observations on the Effects of Betamethasone in Rheumatoid Arthritis. *Ibid.* 10. Cohen, A., and Goldman, J.: Management of Rheumatoid Arthritis with a New Steroid. *Ibid.* 11. Gordon, D. M.: Betamethasone—A New Corticosteroid in Ophthalmology. *Ibid.* 12. Abrahamson, I. A., Jr.: A Clinical Evaluation of Betamethasone. *Ibid.*

H-371

(betamethasone) **Tablets, 0.6 mg.**

CELESTONE™

a new magnitude in corticosteroid activity

the first complete physiologic regulator of female cyclic function

ENOVID®

(brand of norethynodrel with ethynylestradiol 3-methyl ether)

The basic action

ENOVID closely mimics the balanced progestational-estrogenic action of the functioning corpus luteum. This action is readily understood by a simple comparison. In effect, ENOVID *induces a physiologic state which simulates early pregnancy — except that there is no placenta or fetus.* Thus, as in pregnancy, the production or release of pituitary gonadotropin is inhibited and ovulation suspended; a pseudodecidual endometrium ("pseudo" because neither placenta nor fetus is present) is induced and maintained. Further, during ENOVID therapy, certain symptoms typical of normal pregnancy may be noted in some patients, such as nausea — which is usually mild and disappears spontaneously within a few days — breast engorgement, some degree of fluid retention, and often a marked sense of well-being. There is no androgenicity. ENOVID *is as safe as the normal state of pregnancy.*

The basic applications

1. Correction of menstrual dysfunction. *Cyclic* therapy with ENOVID controls dysfunctional uterine bleeding (menorrhagia, metrorrhagia) and often establishes a normal menstrual cycle in amenorrhea.

2. Ovulation suppression (to suspend fertility). For this purpose ENOVID is administered *cyclically,* beginning on day 5 through day 24 (20 daily doses). The ovary remains in a state of physiologic rest and there is no impairment of subsequent fertility. Continuous administration for more than two years is not recommended.

3. Postponement of the menses for reasons of health (impending hospitalization for surgery, during treatment of Bartholin's gland cysts, acute urethritis, rectal abscess, trichomonal or monilial vaginitis), travel, forthcoming marriage, or pressing business or professional engagements. For this purpose ENOVID may be started at any time in the cycle up to one week before expected menstruation. Upon discontinuation, normal cyclic bleeding occurs in three to five days.

4. Threatened abortion. *Continuous* ENOVID treatment provides balanced hormonal support for the endometrium in threatened or habitual abortion.

5. Endocrine infertility. ENOVID has been used successfully in *cyclic* therapy of endocrine infertility, promoting subsequent pregnancy through a probable "rebound" phenomenon.

6. Endometriosis. *Continuous* therapy with ENOVID corrects endometriosis by producing a pseudodecidual reaction with subsequent absorption of aberrant endometrial tissue.

The basic dosage

Basic dosage of ENOVID is 5 mg. daily in *cyclic* therapy, beginning on day 5 through day 24 (20 daily doses). Higher doses may be used with complete safety to prevent or control occasional "spotting" or breakthrough bleeding during ENOVID therapy, or for rapid effect in the emergency treatment of dysfunctional uterine bleeding and threatened abortion.

ENOVID is available in tablets of 5 mg. and 10 mg.

Literature and references, covering over five years of intensive clinical study, available on request.

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...*unfettered*

From the beginning, woman has been a vassal to the temporal demands—and frequently the aberrations—of the cyclic mechanism of her reproductive system. Now, to a degree heretofore unknown, she is permitted normalization, enhancement, or suspension of cyclic function and procreative potential. This new physiologic control is symbolized in an illustration borrowed from ancient Greek mythology—Andromeda freed from her chains.

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Baker's Modified Milk supplies a protein level of 3.7 grams per kilogram of body weight per day to meet normal needs and to provide a dietary reserve for increased protein demands caused by fever, diarrhea and infections. An adequate water reserve is also provided for renal function and proper water balance by the 20 Cal/oz. dilution.[3]

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OPTIMUM NUTRITION
Providing all the normal dietary requirements plus a reserve for stress situations while avoiding the hazards of excessive amounts of individual nutrients.

1. Hill, L. F.: J. Pediatrics 54: 545 (1959) 2. Gordon, H. H., and Ganson, A. F.: J. Pediatrics 54: 503 (1959) 3. A.A.P. Committee on Nutrition, Pediatrics 19: 339 (1957) 4. Woodruff, Calvin W.: J. A. M. A. 175: 114 (1961) 5. Recommended Dietary Allowances, NAS-NRC Publication 589 (1958) 6. U.S.P.H.S. Milk Code, Federal Security Agency Publ. 220 (1953)

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Made only from Grade A milk[6]—scientifically formulated to duplicate the nutritional results of human milk. *Powder/Liquid*

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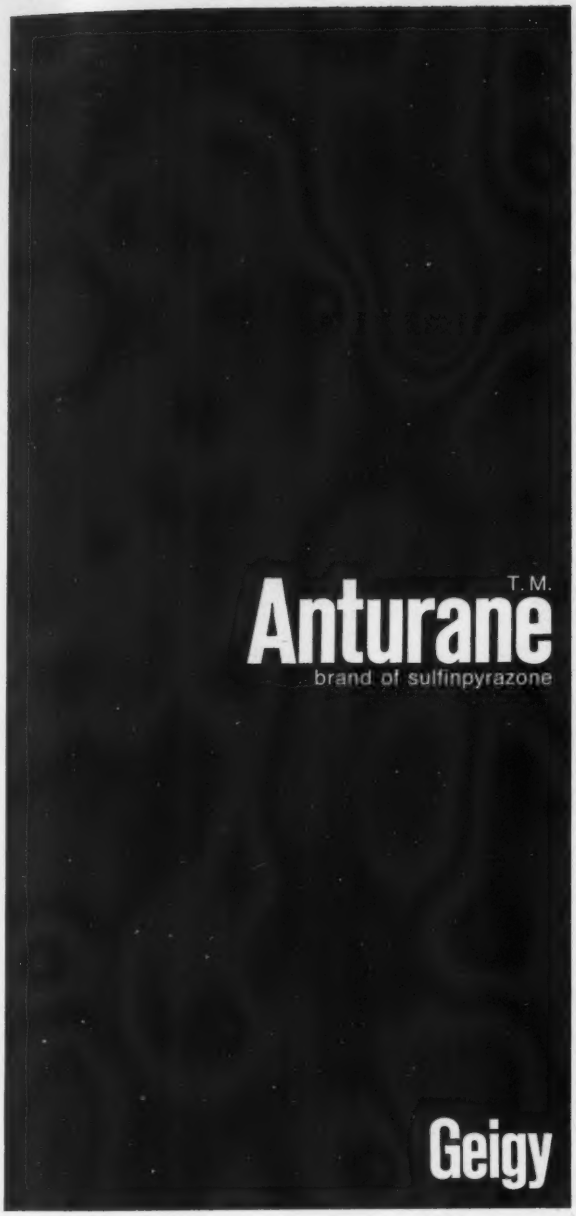

effectively checks the progress of gout by intensive, sustained, uricosuric action

In the long-term treatment of gout, the value of a uricosuric agent such as Anturane in preventing accumulation of the urate deposits responsible for chronic disability and joint derangement is well established.[1]

Clinical experience has demonstrated that Anturane not only prevents formation of new urate deposits, but with sustained therapy, also causes absorption of preexisting tophi.[2] With reduction in hyperuricemia, the incidence and severity of acute attacks are reduced,[3] the mobility of affected joints is improved,[4] and lingering joint pain is relieved.[2,4]

References: 1. Seegmiller, J. E., and Grayzel, A. I.: J.A.M.A. **173**:1076, 1960. 2. Yü, T. F., Burns, J. J., and Gutman, A. B.: Arth. & Rheumat. 1:532, 1958. 3. Kersley, G. D., Cook, E. R., and Tovey, D. C. J.: Ann. Rheumat. Dis. 17:326, 1958. 4. Gutman, A. B., and Yü, T. F.: Bull. New York Acad. Med. 34:287, 1958.

Full product information regarding dosage, side effects, precautions and contraindications available on request.

Anturane[T.M.], brand of sulfinpyrazone: Scored tablets of 100 mg. in bottles of 100.

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Division of Geigy Chemical Corporation
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AM 561-61

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References: 1. Finkel, M. J.: M. Times 86:1391, 1958. 2. Doshay, L. J., and Boshes, L.: Postgrad. Med. 27:602, 1960. 3. A. M. A. Council on Drugs: New and Nonofficial Drugs 1960, Philadelphia, J. B. Lippincott Company, 1960, p. 264.

COGENTIN is a trademark of Merck & Co., Inc.

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A-50

refreshing . . .

delicious . . . stir into water and drink

FOR THE DIFFICULT OR OBSTINATELY CONSTIPATED

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(carboxymethylcellulose with a peristaltic stimulant)

7 Gm. white granular powder contains:
Sodium Carboxymethylcellulose ..... 2 Gm.
Di(acetylhydroxyphenyl)isatin ..... 5 mg.
(chemically similar to the active principle of prunes)

FOR THE CHRONICALLY OR ROUTINELY CONSTIPATED

Effersyl

(psyllium with a mild peristaltic stimulant)

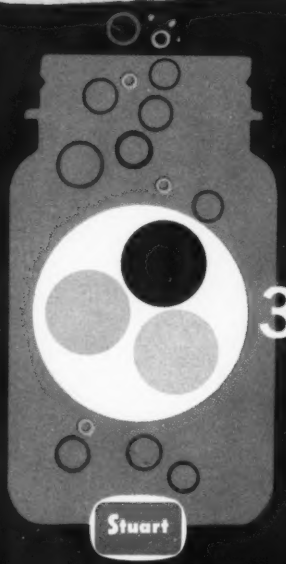
7 Gm. pink granular powder contains:
Psyllium hydrocolloid ..... 3 Gm.
the highly purified hemicellulose of the husk of the psyllium seed (Plantago ovata, Forsk).
Di(acetylhydroxyphenyl)isatin ..... 3 mg.
(chemically similar to the active principle of prunes)

FOR PATIENTS WHO NEED BULK ONLY

Effersyllium

(psyllium with no peristaltic stimulant added)

7 Gm. tan granular powder contains:
Psyllium hydrocolloid ..... 3 Gm.
the highly purified hemicellulose of the husk of the psyllium seed (Plantago ovata, Forsk).



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All three lubricating . . . non-irritating
All three produce soft, well-formed stools.

DOSAGE

Adults: 1 rounded teaspoonful (7 Gm.) in a glass of water daily. Subsequent dosage to be adjusted according to results.

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The whole family will enjoy the three delicious flavors:

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LOW-IN-CALORIES, 10 PER DOSE.

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A	5,000 USP units
D	1,000 USP units
C	75 mg.
B_1	2 mg.
B_2	2 mg.
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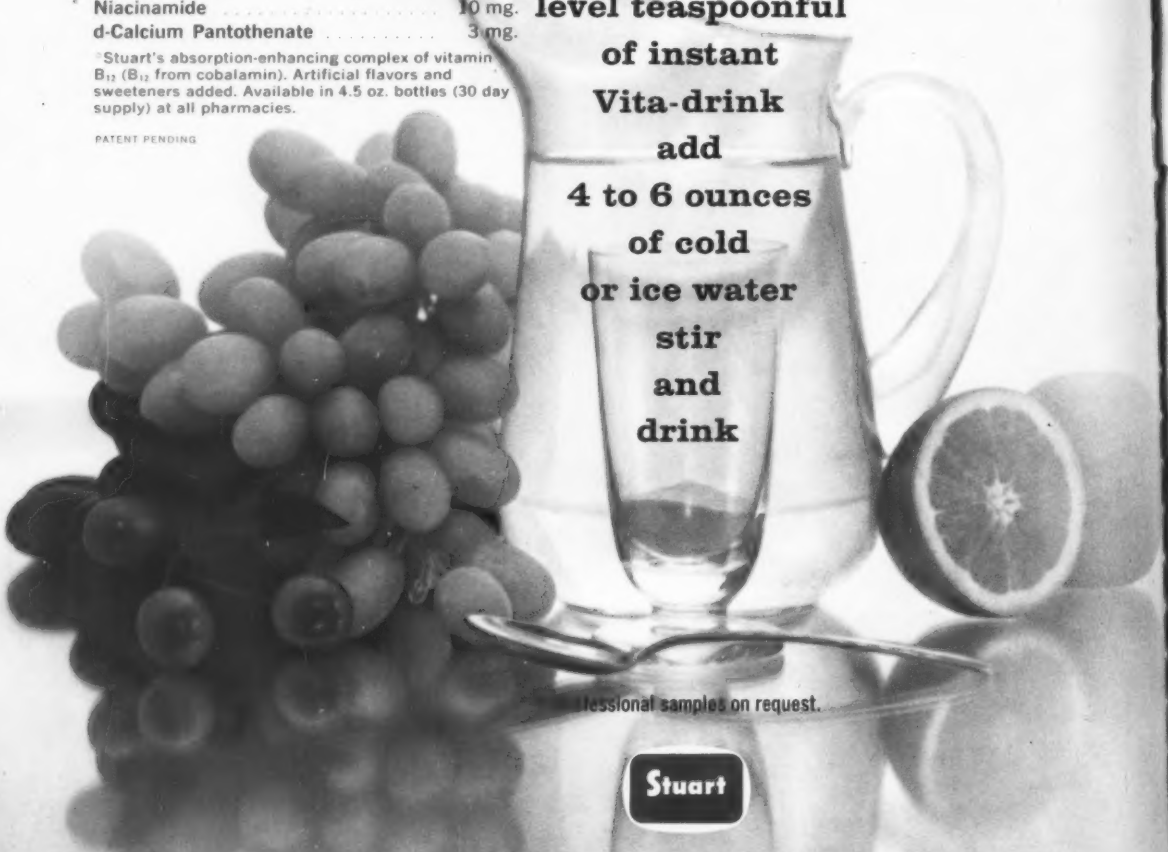

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(1) Boland, E. W.: World-Wide Abstr. Gen. Med. **3**:11, 1960. (2) Lockie, L. M.: Am. J. Orthopedics **2**:252, 1960.

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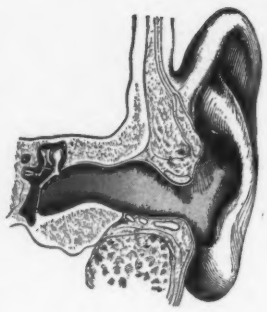

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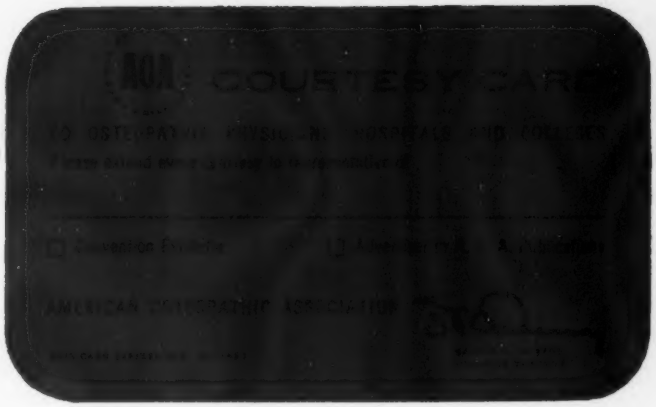

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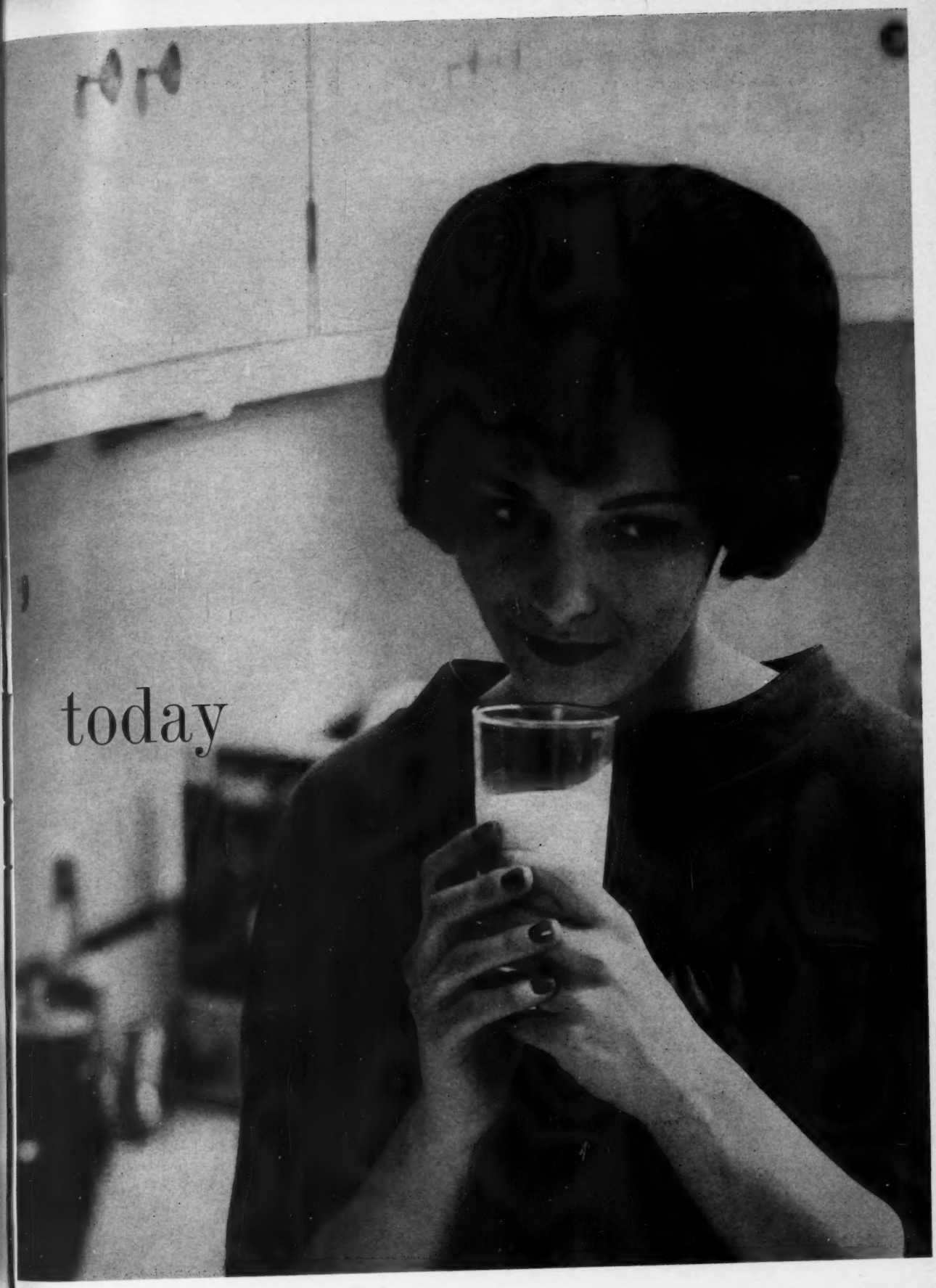
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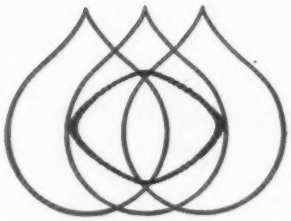

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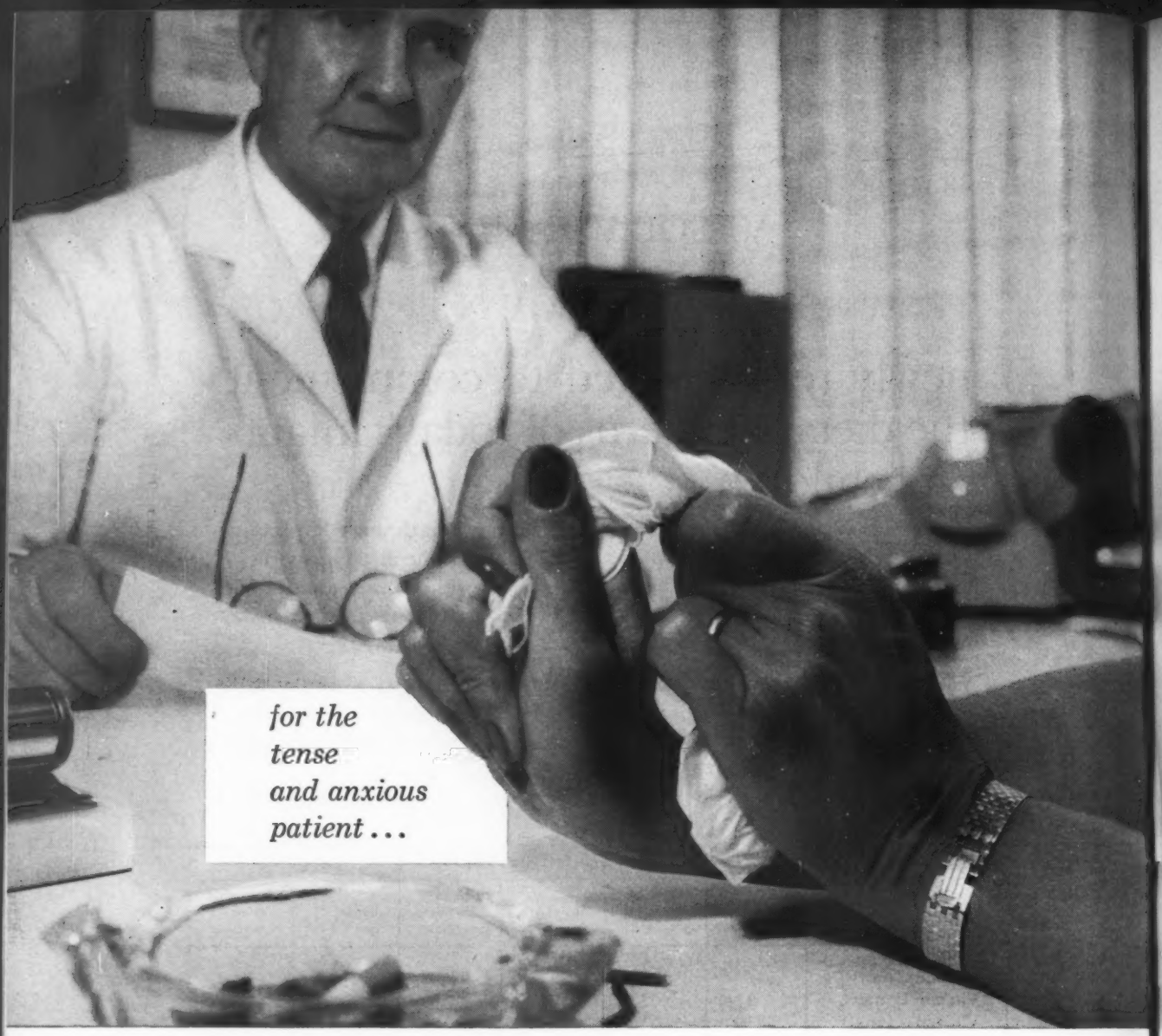

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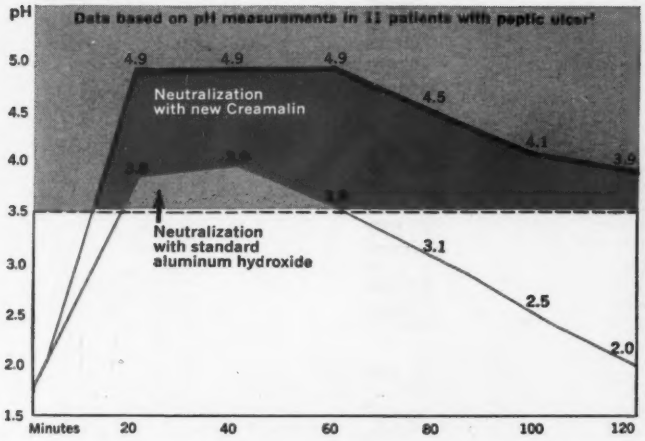

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1. Data in the files of the Department of Medical Research, Winthrop Laboratories. 2. Hinkel, E. T., Jr.; Fisher, M. P., and Tainter, M. L.: J. Am. Pharm. A. (Scient. Ed.) 48:384, July, 1959.

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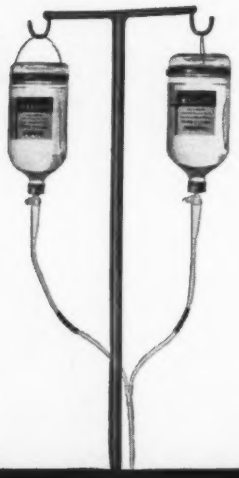

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**Stinebaugh, B. J.: A.M.A. Arch. Int. Med. 105:613, 1960.*

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Manipulative body mechanics therapy: II. Upper dorsal lesion complex

J. WILLARD JOHNSON, D.O., Minneapolis, Minnesota*

The primary lesion in the upper dorsal region is a slip of the costovertebral joint. As with the sacroiliac joint, the lesion apparently involves a traumatically produced abnormal relationship of the articular surfaces which locks the joint against normal free motion.

Costovertebral maladjustment is typically encountered as a group lesion, involving the first and second dorsal costovertebral joints and producing a characteristic pattern of abnormal mechanics in the upper dorsal region. The lesion is apparently predisposed by the normally restricted movement between the ribs and vertebrae in this area.

Costovertebral maladjustment is intimately associated with varying degrees of muscle spasm, inflammation, fibrositis, and arthritis involving the intervertebral structures of the upper dorsal area. The lesion is identified by sensitiveness to pressure, stiffness upon applying springing pressure over the area, and frequently by a marked "hardness" palpated across the upper two or three dorsal transverse processes and ribs. The chronic nature of the lesion, in most instances, suggests injury sustained at an early age. The pathologic changes develop through succeeding years because of strain resulting from the costovertebral maladjustment and from the gradually developing restricted mobility in the area. In a spinal area in which movement is restricted, inability to conform to normal function of the spine in body activity produces irritation and strain in the affected area.

Costovertebral maladjustment and the accompanying upper dorsal intervertebral lesion will be referred to as the "upper dorsal lesion complex."

In children the presence of the upper dorsal lesion complex is the rule rather than the exception. Symptoms of pain are not often present during the early years, but usually develop in one form or another in later life. Somatovisceral reflexes originating in the area produce changes in autonomic function (visceral and circulatory) which are of particular importance because of the segmental association of the upper dorsal region with vital functions.

Symptoms

Symptoms commonly associated with the upper dorsal lesion complex include:

1. Pain and tension in the upper border of the trapezius muscle, extending from the suboccipital or mastoid regions down the side of the neck to the point of the shoulder.
2. Pain and tension in the upper dorsal, cervical, and suboccipital regions, from mild tension to the acute stiff neck.
3. Headache, from the occasional tension headache to the severe migraine. (While there are many precipitating causes of headaches, costovertebral maladjustment is found to be the usual primary, or predisposing, cause.)
4. Symptoms commonly referred to by the patient as "sinus trouble" (suboccipital and supraorbital tension and headache, eye strain, nasal congestion, and postnasal discharge). Dental pain may be greatly aggravated by costovertebral strain.
5. Shoulder pain (referred pain, bursitis, brachial neuritis).
6. Numbness and tingling, rheumatic aches and pains, and arthritis in the fingers, hands, and arms.
7. Vertigo, usually in certain positions or on performing certain movements.

Part I, on the sacroiliac joints, appeared in the June issue of THE JOURNAL.

*Address, 2723 E. 38th St.

In children the presence of an upper dorsal lesion complex is the rule rather than the exception. Symptoms of pain are not often present during the early years, but usually develop in one form or another in later life

8. Ocular disturbances (refraction changes, erratic visual disturbances).
9. Ringing or buzzing in the ears.
10. Tension in the muscles of the throat (sensation of a "lump" in the throat).
11. Habit spasms, particularly in children.
12. Nervous tension and fatigue.

These symptoms may be present singly or in any combination, and with recovery following treatment a recurrence of the lesion may result in the same or a different combination of symptoms.

In cases of migraine headache or epileptiform attacks the upper dorsal lesion complex is usually found to be the determining etiologic factor. Occasionally clinical experience suggests an additional causative factor in the form of nerve damage which sensitizes the patient to symptoms. It seems probable in these cases that the original injury occurred at birth and involved minor nerve damage.

In sprains and fractures involving the upper extremities in which, for no apparent reason, pain and swelling persist or arthritis develops, the cause is usually to be found in circulatory interference resulting from costovertebral maladjustment and strain.

The failure of normal spontaneous recovery in "whiplash" injuries is found to be caused by costovertebral maladjustment. The situation is frequently complicated by the fact that the injury is superimposed upon existing upper dorsal strain in which secondary arthritis has developed, and the injury results in an acute flare-up of the previously chronic, and perhaps subsymptomatic, process.

Somatovisceral reflexes originating in the upper dorsal lesion complex are found to be a most important etiologic factor in thyroid hyperfunction, arterial hypertension, and gastrointestinal dysfunction. The importance of the upper dorsal lesion in coronary heart disease is becoming increasingly apparent.

As with the sacroiliac joint, a costovertebral slip is seldom accompanied by symptoms of pain and discomfort at the time of its occurrence. Symptoms develop gradually as a result of the strain caused by the slip and from the accompanying abnormal mechanics. It is also noted that the pain or discomfort may disappear with no change in the mechanical situation, and then reappear later. It is therefore important that treatment be given on the basis of establishing mechanical normalcy rather than on the basis of symptom response.

Diagnosis of the slipped costovertebral joint

The diagnosis of slipped costovertebral joints is based on mechanical asymmetry, and upon tension and sensitiveness of irritated musculature. The following mechanical changes from normal symmetry are noted:

1. With the patient sitting or standing, head and dorsal spine in forward flexion, the first ribs are palpated (through intervening soft tissue structures) from above downward, and somewhat medially, close to the base of the neck. The rib is elevated on one side (usually the left) and depressed on the other.
2. The second ribs are palpated from above downward, deeply, at a point about halfway between the point of the shoulder and the base of the neck, and between the clavicle and the upper border of the trapezius muscle. The second rib is elevated on the same side and depressed on the other.
3. There is a fullness or prominence over the transverse processes and ribs on the side upon which the ribs are elevated, which reflects rotation of the upper dorsal vertebra toward this side.
4. Rotation of the upper dorsal vertebra carries the ribs forward on the side of the depressed ribs and backward on the other. With the patient in a dorsal position, the second and third ribs are prominent and sensitive and offer greater resistance to springing pressure on the side upon which they are carried forward (usually the right).

The strain produced by slipped first and second dorsal costovertebral joints results in palpatory findings based upon soft tissue changes as follows:

1. There is marked sensitiveness to pressure upon palpating the ribs from above downward.
2. The upper border of the trapezius muscle is tense and sensitive on the side upon which the ribs are elevated.
3. There is tension and sensitiveness in the muscles over the costotransverse articulations and the angles of the ribs.

While the costovertebral joints are usually slipped with the bodies of the ribs elevated on the left side and depressed on the right, the reverse is occasionally encountered and must not be overlooked.

Costovertebral lesion mechanics

In the study of upper dorsal costovertebral mechanics it is significant to note that the first ribs are attached rigidly to the manubrium sterni (syn-

chondroses), so that the first ribs and manubrium move as a single unit. Upon complete inspiration there is slight elevation of the first ribs with corresponding movement at the costovertebral joints ("pump-handle" motion). The second ribs attach directly at the junction of the manubrium with the sternum, but the articulations are diarthrodial in type and permit slight motion. The second ribs thus move with the first ribs upon complete inspiration, and in addition perform restricted movement about an anteroposterior axis in which the bodies of the ribs are elevated ("bucket-bail" motion).

The relative rigidity of the first and second rib mechanism is conducive to slight maladjustment and locking of the costovertebral joints. The lesions occur with elevation of the bodies of the ribs on one side and depression of the bodies of the ribs on the other, and the vertebrae are rotated toward the side upon which the ribs are elevated. Since for the most part the upper two ribs do not normally perform this "bucket-bail" type of motion, costovertebral slips occur, in which the head of the rib apparently slips downward in relation to the vertebra on the side upon which the rib is elevated and upward on the other. This movement of the rib occurs around an axis which passes through the costotransverse joint and the sternal attachment of the rib. The lesions are produced by strained or sudden movements or by strained positions of the head or shoulders.

The above interpretation of the costotransverse and capitular mechanics involved in a costovertebral slip is based upon clinical observation of the effects of various manipulative procedures. Manipulative technic designed according to this mechanism is effective in correcting the lesions. Methods which are counter to the mechanism are usually unsuccessful and, if applied after the lesions have been corrected, will usually cause the lesions to recur.

The mechanical defects encountered in the upper dorsal and lumbopelvic regions are found to conform to a definite pattern with remarkable consistency. Costovertebral slips typically occur with the ribs elevated on the left side and depressed on the right, and with rotation of the upper dorsal vertebrae toward the left. The sacroiliac joints are slipped most frequently in such a way that, with the patient standing in forward flexion, the pelvis and lumbar spine are rotated backward on the right, and the right innominate appears to be rotated backward and the left forward. Differences in leg length complicate but do not materially change this picture.

Treatment of the slipped costovertebral joint

On the side upon which the first and/or second ribs are depressed (usually the right), treatment of the costovertebral slip (head of the rib slipped upward in relation to the body of the vertebra) is carried out as follows (Fig. 1):

1. The patient lies in a ventral position with the point of his chin resting on a small pillow (preferably foam rubber). The head is in complete extension. The arm on the side to be treated rests on the table at the patient's side. The operator stands at the side of the joint to be treated (the side of the depressed rib).

Fig. 1

2. The operator localizes firm counterforce with the hypothenar eminence of one hand over the end of the first and second dorsal transverse processes (over the costotransverse joints), directing the force ventrally and cephalad.

3. The head is rotated toward the opposite side (the occiput moves away from the operator) to the end of the range of relaxed motion, the movement pivoting on the chin. The head rests in this position.

4. The operator bends forward to contact the localizing hand with his lower ribs on the same side.

5. The heel of the free hand is placed high on the patient's forehead, just anterior to the templar region; the elbow is held well out from the trunk and in contact with the table; and the fingers extend back over the top of the occiput.

6. The head is now carried sharply in *extension* and rotation toward the opposite side, against counterforce maintained over the transverse processes in a ventral and cephalad direction. The movement is one of high speed and low excursion, followed by quick release.

The primary force is extension with rotation applied as a locking mechanism. The rotation locking is introduced by the position of the heel of the hand on the anterolateral aspect of the occiput.

The manipulation forces the body of the vertebra upward (extension in relation to the head of the rib), the movement pivoting at the costotransverse joint (the point at which counterforce is localized).

The maneuver is usually effective in correcting slips of both the first and second costovertebral

Fig. 2

joints at the same time. It is noted that the first dorsal transverse process is at the level of the seventh cervical spinous process (vertebra prominens), and the second and third dorsal transverse processes are at the level of the first and second dorsal spinous processes, respectively.

On the side upon which the first and/or second ribs are elevated (usually the left), treatment of the costovertebral slip (head of the rib slipped downward in relation to the body of the vertebra) is performed as follows (Fig. 2):

1. The patient lies in a ventral position with the point of his chin resting on a small pillow. The arm on the side to be treated is dropped down off the table. The operator stands at the side of the joint to be treated (the side of the elevated rib).

2. The operator localizes firm counterforce with the hypothenar eminence over the ends of the first and second dorsal transverse processes (over the costotransverse joints), directing the force ventrally, caudad, and somewhat laterally.

3. The head is rotated toward the opposite side (occiput moves away from the operator) to the end of the range of relaxed motion, the movement pivoting on the chin. The head rests in this position.

4. The operator bends forward to contact the localizing hand with his lower ribs on the same side.

5. The heel of the free hand contacts the occiput just above the ear.

6. The head is now carried sharply in *lateral flexion,* rotation, and extension toward the opposite side, against counterforce maintained over the transverse processes in a ventral, caudad, and somewhat lateral direction. The movement is one of high speed and low excursion, followed by quick release. Rotation of the head is accented as a locking mechanism as the lateral flexion thrust is applied. The rotation is introduced by the position of the heel of the hand well back on the side of the occiput (above the ear).

The manipulation forces the body of the vertebra downward (lateral flexion) in relation to the head of the rib, the movement pivoting at the costotransverse joint. Visualization of costovertebral mechanisms is facilitated with a vertebral segment at hand.

The maneuver is usually effective in correcting slips of both first and second costovertebral joints at the same time.

Both maneuvers are carried out with the operator bending forward in contact with the localizing hand, to assure that they are performed without a counterthrust over the transverse processes, and to protect the operator against slipping his own costovertebral joints as the movement is applied.

Patient relaxation is essential to successful application of the technics. The patient may be instructed to "let the head and shoulders drop down on the table." He may further be instructed to "take a breath and then let it out," and the thrust is applied at the end of expiration. If relaxation is not secured the head may be rotated from side to side in a relaxed rhythmic manner; when relaxation is sensed the thrust is applied without breaking the rhythm of the rocking movement. In the difficult case, the patient may simply be required to relax. Relaxation is not a negative act, but a positive accomplishment requiring a voluntary effort.

If the maneuvers are not successful it is usually because of inadequate rotation locking, or because the patient is not relaxed. If the treatment is not successful there is a sensation of the force "bouncing back" rather than "going through."

Following successful treatment of the costovertebral joints, any maneuver which involves lateral flexion toward the side upon which the ribs were elevated will usually cause the costovertebral slips to recur. Since treatment on the side of the depressed ribs may involve a degree of lateral flexion toward the side of the elevated ribs, treatment is carried out first on the side of the depressed ribs and then on the side of the elevated ribs.

With the patient in a dorsal position, treatment of slipped costovertebral joints is performed as follows (Fig. 3):

1. The patient lies in a dorsal position with his

Fig. 3

arms folded across his chest. The operator stands at the head of the table.

2. The operator places one hand beneath the occiput and raises the patient's head in flexion.

3. With the other hand flexed and the thumb flexed over the forefinger, force is localized with the knuckle of the thumb at the end of the transverse process (beneath the costotransverse joint). The head is then brought back down on the table.

4. The operator steps forward with one foot (on the side to be treated), bends forward, and braces the elbow on this side against his thigh.

5. The operator contacts the patient's upper forehead with his chest, thus holding the patient's head between his chest and the hand beneath the occiput.

6. Pressure is applied with the localizing thumb (elbow braced against the thigh) in a ventral and medial direction to the point of moderate tension. This is accompanied by slight lateral flexion of the head toward the side being treated.

7. A sharp rotation and *extension* movement of the head is now applied by thrusting straight across with the heel of the hand on the posterior occiput, and at the same time a thrust is applied in a ventral and somewhat medial direction with the localizing thumb.

The maneuver is performed in the same manner on both sides. The method is mechanically accurate on the side of the depressed ribs, and is usually successful on the side of the elevated ribs through forcing movement in the slipped joints.

This technic is particularly useful in bedside treatment, and in the treatment of children. In performing the manipulation on children counterforce is applied with the pad of the thumb, hand open, and the thumb held close to the hand for support.

Careful attention to detail in the application of the costovertebral technics is essential.

The reduction of slipped costovertebral joints is accomplished more easily following treatment of upper dorsal muscle spasm and fibrosis (see later).

The pathologic effects of costovertebral maladjustment and strain develop through the stages of muscle spasm, inflammation, fibrositis, and secondary arthritis. Occasionally a subacute arthritic process is activated by the trauma of a corrective manipulation and by the mechanical change, resulting in an acute flare-up of inflammation and pain. Heat, rest, and gentle manipulation are helpful in this situation. If the costovertebral slips recur they must be corrected with as little trauma as possible. Active arthritis in the upper dorsal region may be suspected in cases in which fibrositis is well developed, and stiffness, "hardness," and sensitiveness to pressure are marked.

While the reduction of costovertebral slips is ordinarily accompanied by some degree of joint "popping," reduction may occur with no audible evidence of joint movement, particularly at a second attempt. Whether or not "popping" occurs thus has no significance with regard to whether or not a manipulation has been successful. The result of treatment must be determined by careful examination.

Since the basic causes of disease may be studied in each patient, and useful conclusions arrived at long before symptoms develop, it becomes possible to institute therapy long before a specific disease syndrome appears

Diagnosis following treatment

The effectiveness of the manipulations is checked by the change toward normal in the mechanical asymmetry, and by the release of muscle tensions and decreased sensitiveness. The examination must be carefully performed. The general pattern of the mechanics of the upper back has usually been established over a period of years; while correction of the primary costal maladjustments improves the over-all picture, the original pattern may still be more or less in evidence and must not confuse the diagnosis. The immediate release of muscular tensions and disappearance of acute sensitiveness upon correction of rib maladjustments are dramatic.

With the patient sitting or standing, the first and second ribs are palpated carefully from above downward. If the manipulative procedure has been successful, a symmetrical relationship is noted. If asymmetry persists, the side upon which treatment was unsuccessful is indicated by *acute* sensitiveness to pressure upon palpating the ribs from above downward, and by the muscle spasm, acute sensitiveness, and "hardness" over the angles of the ribs. Upon release of the rib locking, the acute sensitiveness and tension disappear almost immediately. Examination of the ribs and of the costovertebral areas may be carried out both from in back and from in front of the patient. Accuracy is developed by experience in before-and-after diagnosis.

The fullness, or prominence, over the transverse processes and angles of the ribs on the side upon which the ribs are elevated disappears or is notably diminished with reduction of the costovertebral lesions.

Diagnostic accuracy will be found to depend to a great extent upon the degree of success with which the therapeutic technics are applied. Since both sacroiliac and costovertebral strain are present

Fig. 4

in most individuals, it is necessary that a normal condition of the joints be produced by treatment in order to establish a comparison by which the existence, characteristics, and significance of the abnormal can be identified.

The upper dorsal intervertebral lesion

Costovertebral maladjustment is intimately associated with muscle spasm, fibrositis, and degenerative changes involving the intervertebral structures of the upper dorsal area. Upon palpation the paravertebral muscles feel tense and "ropy," and are sensitive to pressure; the area feels hard and nonresilient according to the extent of the fibrosis and degenerative changes. In cases in which fibrositis is well advanced, a marked "hardness" is palpated across the upper two or three dorsal transverse processes and ribs, and there is usually a degree of increased flexion in the area (kyphotic defect). Secondary arthritis is often well developed in these cases and must be given due consideration in treatment.

In the lower upper dorsal segments and in the middorsal region, a "hyperextension" area is frequently encountered, in which the spinous processes are depressed and approximated each to the other. The spinous process, or processes, at the center of the extended area are acutely sensitive to pressure, reflecting the acute strain and inflammation caused by restricted mobility. The lesion, as above, involves fibrositis and degenerative changes affecting the deep muscles and ligaments and the intervertebral fibrocartilages. The spinous processes are best palpated with the patient in the dorsal position and the head raised in flexion.

In view of the development early in life of the upper dorsal lesion complex and of sacroiliac maladjustment and strain, the common presence of degenerative changes in the intervertebral disks in the young adult is given a basis in long-continued trauma.

Upper dorsal fibrositis and degenerative changes tend to maintain the abnormal vertebral and costal relationships produced by slipped costovertebral joints. The stiffness in the area and the mechanical defect predispose to recurring slips of the joints, and the strain which is inherent in an area of restricted mobility is found to be an important factor in producing the symptoms which are characteristic of the upper dorsal lesion complex.

Treatment of fibrosis and degenerative changes in the upper and middle dorsal areas is performed as follows (Fig. 4):

1. The patient lies in a dorsal position. The operator stands at the side to be treated.
2. A sponge rubber ball 3 inches in diameter is placed beneath the transverse processes on the near side.
3. The arm on the side under which the ball is placed is brought to approximately a vertical position, elbow *extended* and hand closed. (The arm should be at slightly less than a right angle with the trunk.)
4. The other hand is placed back of the head, and the head is raised in complete flexion.
5. The patient is now instructed to "swing the extended arm back as far as he can." This movement is repeated in rhythmic fashion several times, and the patient is then instructed to "rock backward over the ball as the arm swings back." The backward movement of the arm and trunk must end with a relaxed "whip" effect to impose a hyperextension thrust at the point of ball contact. The head is maintained in complete flexion as the movement is performed. The movement is repeated five or six times.

The maneuver is performed in the same manner on both sides, and treatment is applied through the upper and middle dorsal areas as indicated.

In the presence of shoulder pain of such intensity that the patient cannot bring the arm back, the manipulation may be performed with the hands clasped high on the posterior occiput. The head is flexed on the chest, and the patient rocks forward and backward over the ball.

The maneuver exerts a powerful torsion-extension effect between the vertebrae, which forcibly stretches the deep muscles and ligaments and the intervertebral fibrocartilages, breaking down fibrosis and restoring mobility, and thus enabling regeneration of the structures toward normal. As the fibrosis is overcome sensitiveness at the point of ball contact disappears. Muscle tension and "hardness" in the upper dorsal area disappear, flexibility improves, the residual mechanical defect is diminished or removed, and stability of the costovertebral joints (resistance to recurring slips) is increased.

The reduction of slipped costovertebral joints is accomplished more easily following treatment by this method.

It is noted that performance of the manipulation with the head resting on the table instead of with the head held in a fully flexed position will usually cause costovertebral slips to recur.

It is often desirable to have the patient use this

technic at home. The maneuver is performed on the floor in the manner described. The patient may roll on the ball in adjusting it from one position to another. The lacquered finish may be removed from the ball to prevent it from slipping upward.

It is noted that the effects of strain upon the articular structures develop differently in the upper dorsal and in the lower lumbar and upper cervical areas. The normally restricted mobility of the upper dorsal area strongly favors the development of restricting fibrosis, whereas the free mobility of the lower lumbar and upper cervical areas, and the extensive functional requirement in these areas in weight-bearing, favor acute inflammation and muscle spasm and tend to prevent the development of fibrosis. Restricted mobility, "hardness," and nonresilience are thus prominent in the upper dorsal region, and are not often encountered to an important degree in the lower lumbar and upper cervical regions.

Treatment of the lesion mechanism

The mechanism of costovertebral maladjustment involves depression of the ribs on one side and elevation of the ribs on the other, with slight lateral flexion and rotation of the vertebrae toward the side of the elevated ribs. Upper dorsal fibrosis and degenerative changes tend to maintain this mechanical defect. Following reduction of the costovertebral slips this lesion mechanism may be reversed, and stretching treatment applied to the soft tissue structures, by a method which applies lateral flexion and rotation of the vertebrae toward the side of the depressed ribs, as follows (Fig. 5):

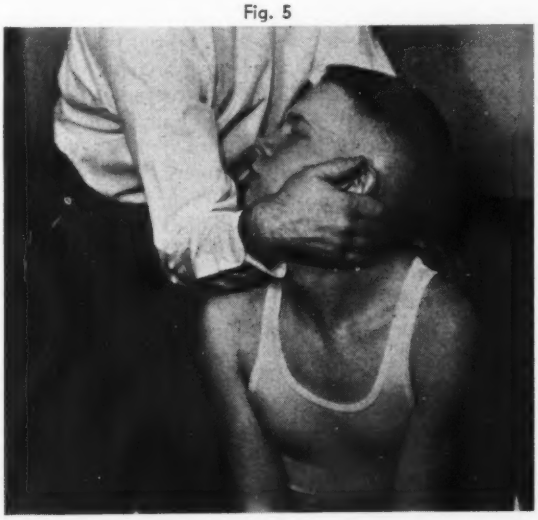
1. The patient is seated on a chair, sitting up straight with head up and arms hanging loosely. The operator stands at the side upon which the ribs were depressed (usually the right).

2. The operator passes one hand in front of the patient's chin, cradling the chin in the angle of his flexed wrist, and placing the hand on the opposite side of the face. The forearm contacts the point of the shoulder.

3. Counterforce is localized with the other hand over the transverse processes on the opposite side of the spine, or against the spinous processes on the near side.

4. Rotation and lateral flexion of the head and upper spine are now alternately applied and released in a springing manner, pulling on the face, chin, and shoulder and pushing with the localizing hand. The level of the localizing pressure may be varied to include the upper and middle dorsal areas.

Fig. 5



Recurring slips of the costovertebral joints

As with the sacroiliac joints, stability of the upper dorsal costovertebral joints increases gradually as mechanical normalcy in the upper dorsal area is restored.

Since slips of the costovertebral joints are seldom accompanied by identifying symptoms at the time of their occurrence, it is usually difficult to determine the exact manner in which the lesions were produced. While the lesions may result from traumatic movements of the head or shoulders, certain positions habitually assumed have been identified, which are found to be of greater importance than direct trauma in causing recurrences of the lesions. The following positions are found to cause costovertebral joints to slip:

1. Lying on the side, the arm on the underside held more or less straight out from the shoulder, and the upper shoulder rolled forward to a position of tension. In this position the hand may be beneath the head or pillow or around the neck, or the forearm may be extended straight out.

2. Lying on the side with the arm on the underside extending upward and the head resting thereon.

3. Lying on the side with the arm on the underside extending straight downward, and the trunk rolled forward onto the arm.

4. Lying on the back or side with the head propped up toward a vertical position. This position is usually assumed in reading while lying down.

5. Lying in the ventral position with the head turned sharply sideways.

6. Straining to reach in any direction.

Regarding positions one, two, and three the patient may be instructed, "When lying on the side keep the arms down, and do not let the upper shoulder roll forward."

Since both the sacroiliac and costovertebral joints are readily slipped while lying in the ventral position, sleeping in this position should be discouraged.

Reduction of recurring slips of the costovertebral joints may frequently be accomplished without the assistance of an operator, by the following maneuvers:

The depressed rib technic (right side), is as follows:

Fig. 6

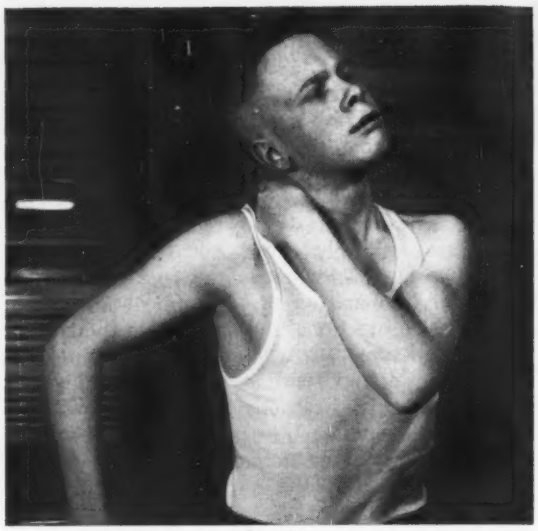


Fig. 7

1. The maneuver is performed in the standing position.

2. Using the third and fourth fingers of the left hand (little finger flexed on the hand) firm pressure is applied over the right first and second costotransverse joints in a forward and downward direction. The pressure must be firm, and localized accurately over the costotransverse joints (over the ends of the transverse processes). The location of the first dorsal transverse process at the level of the seventh cervical spinous process must be kept in mind.

3. The head is extended slightly and tilted toward the left side (Fig. 6).

4. A quick movement of the right shoulder and head is now performed, in which the shoulder is elevated sharply and the head is thrown in right lateral flexion and rotation toward the left (posterior occiput moves toward the point of the right shoulder). The movement of the shoulder is reinforced by bringing the arm up sharply in *abduction*, flexing the elbow (Fig. 7).

The elevated rib technic (left side) is as follows:

1. The maneuver is performed in the standing position.

2. Using the third and fourth fingers of the right hand, firm pressure is applied over the left first and second costotransverse joints in a forward and downward direction.

3. The left shoulder is elevated, the elbow is flexed somewhat, and the head is extended slightly and tilted toward the left side. The position is maintained in a relaxed manner (Fig. 8).

4. A sudden movement is then performed, in which the left hand and shoulder thrust sharply downward, and the head is thrown in right lateral flexion and rotation toward the left (posterior occiput moves toward the point of the right shoulder) (Fig. 9).

The maneuvers are performed in a relaxed, sudden manner to produce a sharp thrust at the point at which force is localized with the fingers. The corrective force is developed mainly by the movement of the shoulder. An attempt is made to localize the lateral flexion movement of the head at the point at which force is localized with the fingers. It is noted that the movement of the head is in the same direction in both technics.

The technics are mechanically accurate. Practice is required in order to achieve the synchronization of movement necessary to successful performance of the maneuvers. Diagnosis in self-treatment is based upon symptoms, and upon examination of the rib areas in front of a mirror.

Manipulative body mechanics therapy: summary

The essential basic lesions which produce strain and irritation in body mechanics are slipped sacroiliac joints, slipped upper dorsal costovertebral joints, fibrosis which restricts mobility of the upper dorsal spinal segments, and body imbalance (mainly unequal leg length). Effective treatment of these basic lesions is for the most part clinically adequate in body mechanics therapy. With removal of basic lesions secondary muscle spasm and inflammation resolve spontaneously. Secondary fibrositis and arthritis may require direct manipulative treatment to improve mobility of the involved joints, and to improve circulation.

The manipulative treatment of reflex spinal lesions is a separate field of application of manipulative science.

An approach to therapy

Man may be defined as a mechanically and chemically functioning organism, reacting to his environment according to his intellectual and emotional

Fig. 8

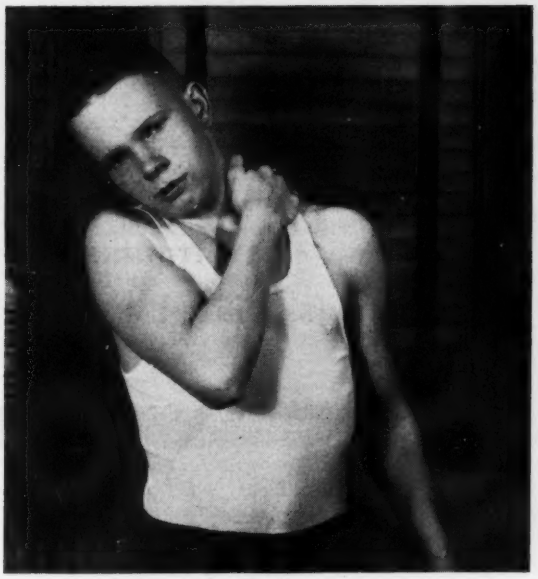


Fig. 9

capacities, and subject to inherent characteristics of function and to the external influences of his environment. The basic causes of disease may thus be outlined as: (1) mechanical stress, (2) nutritional excess or deficiency (chemical imbalance), (3) emotional trauma, (4) unhygienic habits of living, (5) inherent predispositions to disease, and (6) external injury.

The osteopathic concept of medicine, which attaches greatest importance to treatment of the patient rather than of the disease, approaches diagnosis from the point of view of determining primarily what is wrong with the patient that he has the disease, and then directs treatment toward removal of these underlying causes. The disease entity which classification of his symptoms represents is considered for the benefit of whatever specific therapy may be available for that particular disease.

Since the basic causes of disease may be studied in each patient, and useful conclusions arrived at long before symptoms develop, it becomes possible to institute therapy long before a specific disease syndrome, with well developed pathologic changes, appears. Therapy is thus oriented to health maintenance rather than to the treatment of disease.

This approach to therapy becomes practical to a very great extent because of the major importance of mechanical stress as a cause of disease, and because of the possibility of the control of mechanical stress through manipulative body mechanics therapy.

Prevention and minimization of barotraumatic injuries associated with scuba diving

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Scuba diving is becoming an increasingly popular sport. In such an unlikely geographic situation as St. Louis, Missouri, over 1,200 persons have taken formal courses in scuba (self-contained underwater breathing apparatus—the "aqualung") diving. In this city alone, there are at least five formal organizations of enthusiasts, and one local supplier reports the sale of over 200 instruments for prolonged submersion. Duffner,[1] in his recent treatise, describes the increased interest in underwater recreation with precision and eloquence.

Recently, the medical literature has shown cognizance of the special injuries suffered by scuba enthusiasts.[1-3] These injuries, once considered remote and exotic entities, have become commonplace in many parts of the country and are seen frequently by the family physician. When one considers that a million "aqualungs" have been sold in this country, it becomes apparent that their use will increase the incidence of underwater injuries. Most physicians are aware of these hazards, but many are unsure of their own connection with the prevention, causation, treatment, and sequelae of such injuries.

Trauma associated with scuba diving may be categorized as problems of descent, of prolonged submersion, and of ascent.[1-3] The descending and ascending diver is exposed to rapid changes in pressure. With every foot of descent, the pressure increases 0.445 psi (pounds per square inch), and the reverse is true on ascent. This means that the pressure of the respiratory mixture must change rapidly to permit an equalization of the pressure in the air-containing spaces of the body with that of the surrounding water. These spaces include the nasal air passages, lungs, paranasal sinuses, and middle ears. Even the minute air pockets beneath faulty dental fillings and inlays become important. If this equalization is not accomplished by air-pressure changes, pressures on surrounding body fluids and tissues will come into play. When these changes are sufficiently rapid to create a marked pressure differential, the tissues themselves will rupture. "Aqualungs" are designed to compensate for pressure changes by automatic adjustment of the pressure on the air mixture according to the depth of submersion. Obviously, since this is a mechanical device, there are certain inherent limitations to the system, and the physiologic mechanisms of the body must effect the final gaseous equilibrium.

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Problems of descent

Aerotitis media • As the scuba diver descends, water pressure on the outer ear must be equalized by the pressure in the middle ear. This occurs naturally if the eustachian tubes remain patent and allow free interchange of pressure potentials between the middle ear and the nasopharynx. If these canals are not patent, the hazard is obvious. As the depth of submersion increases, the pressure differential increases. When it exceeds the critical tissue tolerance (an idiopathic and variable value), a pathologic response occurs. These changes vary from microhemorrhages in the tympanic membrane to frank middle ear hemorrhage with obliteration of the air space. Teed has classified this injury by gradations of one to five, depending on the magnitude of objective findings.[2]

Although the mechanism of trauma is identical to that experienced in aviation (aerotitis media), the frequency of occurrence and the severity of symptoms, findings, and sequelae are not parallel. Since these cannot be regarded as the same diseases, the

term "barotitis media" is proposed to specify the injury sustained in water. Further subclassifications, "barotitis media descendans" and "barotitis media ascendans," are suggested, depending on whether the trauma occurs during descent or ascent.

When a diving injury results in the obliteration of the middle ear cavity with blood, the diver may not be aware of the trauma; he may continue a comfortable descent, and he may be asymptomatic for hours after the termination of the diving period. He will complain of a feeling of fulness in the involved ear, diminished auditory acuity, and possibly an audible splash when the head is moved. He will usually report unusual difficulty in "popping" his ears during descent. The vigorous blowing frequently results in infection of the middle ear. Otoscopic findings of extravasated blood behind the tympanic membrane confirm the diagnosis. Frequently a fluid level is demonstrable, and occasionally some of the blood may escape through the eustachian tubes into the oropharynx and be noted in the sputum.

Catheterization of the eustachian tubes should never be undertaken in the treatment of barotitis media descendans even though this procedure affords symptomatic relief. Ear drops generally serve no purpose, either. The treatment of choice is a combination of vasoconstrictors, antibiotics, and enzymatic anti-inflammatory drugs. Since beginning the routine use of antibiotics, I have not encountered abscess of the middle ear. The prognosis is good and permanent sequelae are rare. Complicating infections and repeated trauma are to be avoided for fear of irreparable damage to the middle ear. Prevention, of course, is the "therapy" of choice.

Barosinusitis • The mechanism of this barotraumatic entity is identical to that causing barotitis media descendans. Exudation and hemorrhage into the paranasal sinuses is an extremely common occurrence. The pain caused by the pressure differential in the paranasal sinuses, however, will halt the descent and thereby limit the degree of damage. A roentgen study will demonstrate the opacification of the affected air cells. The treatment is the same as outlined for barotitis media.

Dental pain • This is occasioned by minute, closed air spaces under dental fillings. The condition can be quite painful, but it is never serious. It may persist for some time after the onset and may require analgesics. Repair of the offending tooth is a permanent cure.

Otitis externa • This is not a function of barotrauma but is submersion-linked in its causation. It is identical to the "swimming pool ear" seen frequently in the summer months, and the treatment is the same.

Vertigo • During the descent of a diver whose eustachian tubes are not patent, the tympanic membrane is exposed to considerable force and occasionally may perforate. If the water is cold, labyrinthine disturbances may induce a severe vertigo. The vertigo in itself is not dangerous, but it may result in drowning because of other factors frequently associated with labyrinthine disturbances. These include excessive perspiration, tachycardia, nausea, vomiting, and lowering of blood pressure. Emesis under water is a hazardous undertaking and requires a special technic. Well-trained and experienced divers can accomplish this without seriously interfering with respiration or causing equipment malfunction.

The disorientation accompanying vertigo can become so severe that the diver is unable to determine surface direction, and he will stumble about or cling to a rock until his air supply is exhausted and asphyxiation is inevitable.

Since vertigo is a condition which requires immediate attention and occurs under water where such help is not available, its prevention is of primary concern. Proper instruction and early recognition of the condition are important in minimizing the dangers associated with it.

Subconjunctival hemorrhage • The facial area encompassed by the face mask becomes an air space and depends upon patency of the nasal air passages for pressure equalization. If the diver, through bad habit, closes these passages by valving the nasopharynx with the dorsum of the tongue, a relative negative pressure is created. The subconjunctival capillaries rupture, and hemorrhage follows.

Besides subconjunctival hemorrhage, retrobulbar hemorrhage is also possible; however, I have not observed such a case. The treatment is to hasten reabsorption with anti-inflammatory enzymes. Prognosis is good and serious sequelae have not been observed. This problem can usually be prevented by proper breathing instruction.

Oxygen and carbon dioxide intoxication • Intoxication by these gases is a problem of overpressure-breathing, and will not be considered here. They are both hazards attendant upon special types of apparatus not accessible to the ordinary scuba diver. The use of compressed air generally obviates this problem; however, it is to be noted that oxygen intoxication may occur at depths in excess of 260 feet, even if compressed air is breathed (see the chart).

Nitrogen narcosis—"rapture of the deep" (problems of prolonged submersion) • This strange phenomenon is caused by oversaturation of plasma and tissues of the central nervous system with nitrogen. The narcotic action of nitrogen becomes apparent at depths of about 100 feet as a result of increased partial pressure. This phenomenon resembles in many ways the state of light anesthesia. There are no permanent medical sequelae, and the only danger is that the person will harm himself

because of his distorted judgment and impaired sensibilities. Prevention consists primarily of early recognition of narcosis.

Problems of ascent

Barotitis media ascendans • The air breathed by the scuba diver is extremely dry and acts as a dessicant on the oropharynx. If this continues, the mucus becomes viscous and effectively plugs the eustachian tubes. Then, the exact reverse of descent trauma occurs. As the diver ascends, the expanding gas volume entrapped in the middle ear exerts an outward pressure on the tympanic membrane. The presenting symptoms are pain and a sensation of fullness in the affected ear. The membrane appears convex, dusky in color, and lusterless. The patient is clearly aware that the insult occurred during ascent.

Barotitis media ascendans is experienced to some degree by most divers. The mild discomfort may persist for several hours. Repeated yawning, chewing, and swallowing generally permit escape of enough gas to minimize symptoms. However, in severe cases, catheterization of the eustachian tube affords prompt relief (note that this procedure is contraindicated in barotitis media descendans). Nasal decongestants may likewise be used if wished.

Barosinusitis • If the increase in pressure within the middle ear can cause damage, one would expect that pressure within the paranasal sinuses could also develop. However, no references in the literature have been found, and I have never encountered such a case.

Rupture of pulmonary alveoli • This is probably the most dreaded of all barotraumatic accidents. The following description may illustrate the physiologic changes.[3] If a diver swimming at a depth of 125 feet inhales, holds his breath and ascends, when he reaches the surface, the pulmonary gas content will have increased its volume fivefold. If the diver holds his breath and ascends from 33 feet, the volume will have doubled. If one is aware that pressures of 100 mg. Hg above ambient pressure are sufficient to cause alveolar rupture, it is not difficult to imagine this eventuation. What becomes of the diver after this catastrophe is a function of the volume of the air inocula and the structures to which it gains access.

When gas gains entry to the pulmonary arterial system through the rupture of pulmonary alveoli, it acts as an embolus and travels through the left atrium and ventricle, and often into the brain via the carotid arteries. The embolus may cause cardiac arrest or, less seriously, it may find its way into the capillary system of any viscus. A lethal result is assured if the air inocula is in excess of 300 cc. at the depth of occurrence. The treatment for the fortunate patient who survives is to lay him on his left side in Trendelenburg's position, and get him into a recompression chamber as quickly as possible. Prevention of this catastrophe is possible by emphasizing the cardinal rule that a diver must exhale constantly while in ascent.

If the escaped air gains access to the interstitial pulmonary spaces, it dissects along vascular routes and fascial planes into the mediastinum and into the chest wall and neck. Aeromediastinitis and subcutaneous emphysema are not usually fatal, and the therapy is supportive and palliative. Chest pain is the chief complaint. Crepitus is noticeable if subcutaneous emphysema occurs.

Alveolar rupture into the pleural space causes total or subtotal pneumothorax. Rupture of a bronchopulmonary segment may result from air trapped by a broncholith of dried secretion, and it causes aeromediastinitis, atelectasis, or, conceivably, pneumothorax. Rupture of an emphysematous bleb usually admits air to the pleural space and permits some degree of pneumothorax. Embolization of air or fascial plane dissection may also occur. The treatment is the same as for any other pneumothorax.

It should be mentioned that any or all of these mechanisms can occur simultaneously. From the foregoing, it becomes evident that any disorder that might conceivably produce or allow air entrapment should eliminate the person from barotrauma-producing situations. A rationale of prevention will be dealt with in a later section.

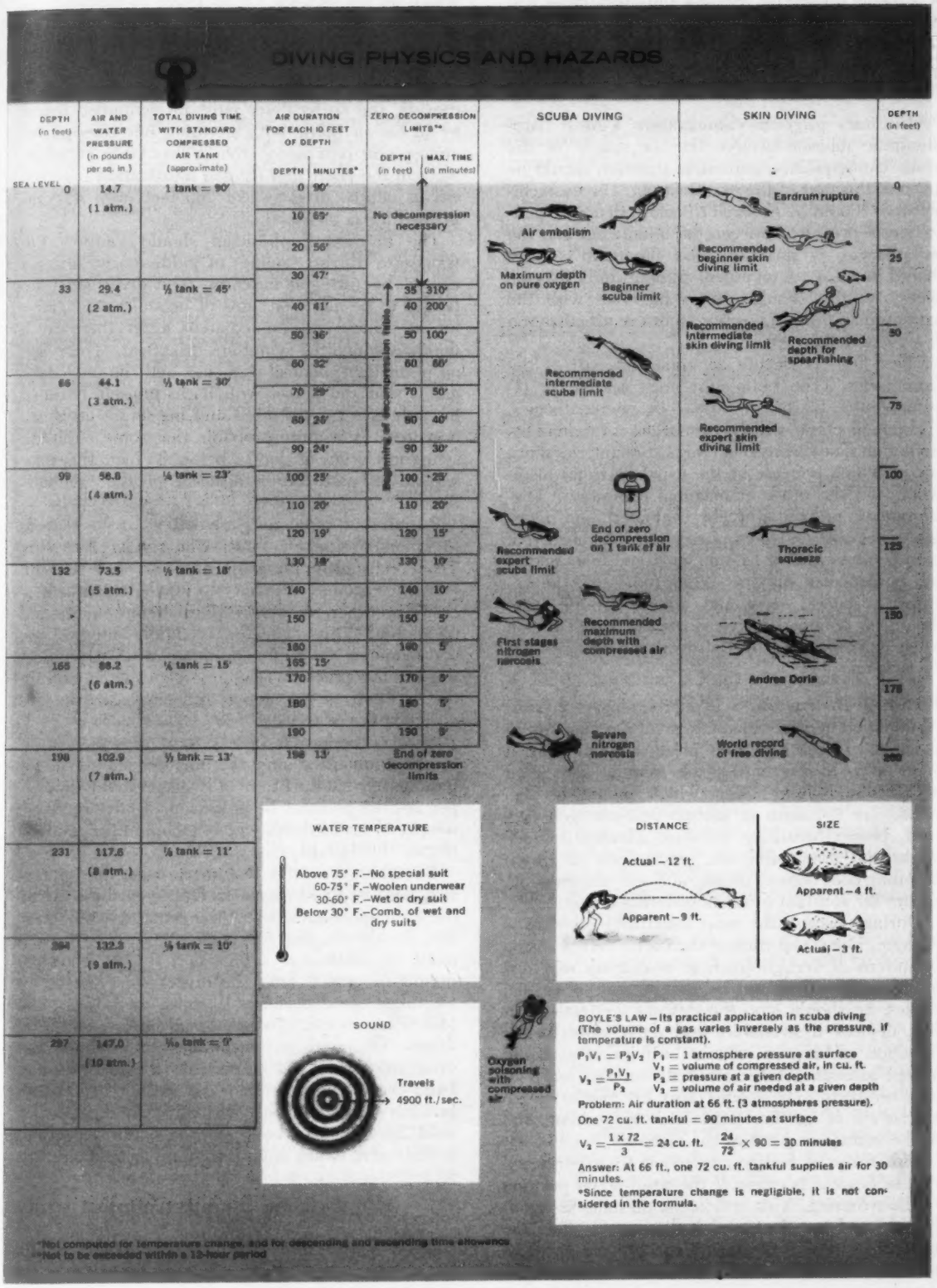
"The bends"—decompression sickness • Nitrogen oversaturation is the etiologic factor in this widely recognized diving illness. It is caused by sudden decompression after overcritical nitrogen saturation. Bubbles of this gas form in the plasma and behave as air emboli. The severity of the symptoms depends on the number of bubbles, their location, and the rapidity of their formation. The most common symptoms are severe joint pain, abdominal cramping, visual disturbances, and paralysis. Central nervous system symptoms may become irreversible with ensuing and irreparable loss of sight or paralysis.

The treatment is immediate recompression followed by gradual decompression. Prevention of "the bends" is a matter of strict adherence to the decompression tables available to any scuba diver where he bought his equipment.

Role of the physician in scuba diving

Most sports today require that participants undergo a fitness examination. Scuba organizations are also beginning to advocate this safety measure. Physicians most likely to be consulted to evaluate candidates for this growing sport are general practitioners and internists. In addition to the general examination, physicians will be expected to detect persons who are unfit to tolerate barotraumatic conditions, and they will have to be familiar with methods of treating injuries resulting from such conditions. Although specialists in eye, ear, nose, and throat diseases will be confronted by a majority

TABLE I*


DIVING PHYSICS AND HAZARDS
DEPTH (in feet)
AIR AND WATER PRESSURE (in pounds per sq. in.)
TOTAL DIVING TIME WITH STANDARD COMPRESSED AIR TANK (approximate)
AIR DURATION FOR EACH 10 FEET OF DEPTH
DEPTH
MINUTES*
ZERO DECOMPRESSION LIMITS**
DEPTH (in feet)
MAX. TIME (in minutes)
SEA LEVEL 0 14.7 (1 atm.) 1 tank = 90'
33 29.4 (2 atm.) ½ tank = 45'
66 44.1 (3 atm.) ⅓ tank = 30'
99 58.8 (4 atm.) ¼ tank = 23'
132 73.5 (5 atm.) ⅕ tank = 18'
165 88.2 (6 atm.) ⅙ tank = 15'
198 102.9 (7 atm.) ⅐ tank = 13'
231 117.6 (8 atm.) ⅛ tank = 11'
264 132.3 (9 atm.) ⅑ tank = 10'
297 147.0 (10 atm.) 1/10 tank = 9'
No decompression necessary
Beginning of decompression table
End of zero decompression limits
SCUBA DIVING
Air embolism
Maximum depth on pure oxygen
Beginner scuba limit
Recommended intermediate scuba limit
Recommended expert scuba limit
End of zero decompression on 1 tank of air
First stages nitrogen narcosis
Recommended maximum depth with compressed air
Severe nitrogen narcosis
Oxygen poisoning with compressed air
SKIN DIVING
Eardrum rupture
Recommended beginner skin diving limit
Recommended intermediate skin diving limit
Recommended depth for spearfishing
Recommended expert skin diving limit
Thoracic squeeze
Andrea Doria
World record of free diving
DEPTH (in feet)
0
25
50
75
100
125
150
175
200
WATER TEMPERATURE
Above 75° F.—No special suit
60-75° F.—Woolen underwear
30-60° F.—Wet or dry suit
Below 30° F.—Comb. of wet and dry suits
DISTANCE
Actual—12 ft.
Apparent—9 ft.
SIZE
Apparent—4 ft.
Actual—3 ft.
SOUND
Travels 4900 ft./sec.
BOYLE'S LAW—Its practical application in scuba diving
(The volume of a gas varies inversely as the pressure, if temperature is constant).
$P_1V_1 = P_2V_2$ P_1 = 1 atmosphere pressure at surface
$V_2 = \frac{P_1V_1}{P_2}$ V_1 = volume of compressed air, in cu. ft.
P_2 = pressure at a given depth
V_2 = volume of air needed at a given depth
Problem: Air duration at 66 ft. (3 atmospheres pressure).
One 72 cu. ft. tankful = 90 minutes at surface
$V_2 = \frac{1 \times 72}{3} = 24$ cu. ft. $\frac{24}{72} \times 90 = 30$ minutes
Answer: At 66 ft., one 72 cu. ft. tankful supplies air for 30 minutes.
*Since temperature change is negligible, it is not considered in the formula.
*Not computed for temperature change, and for descending and ascending time allowance.
**Not to be exceeded within a 12-hour period

*Reproduced by permission of Pfizer Laboratories. The value of 25 minutes opposite 100 feet in the fourth column from the left is incorrect. The time should be 22.4 minutes.

of injured patients, it seems that the responsibility of teaching preventive technics and precautions will rest on the general physician, preferably during the preliminary examination. Therefore, he should acquaint himself with the physics and physiopathology of this new sport.

Preliminary physical examination • Most barotraumatic injuries involve the air spaces of the head. Consequently, particular attention should be given to this part of the examination. The eustachian tubes should be checked to insure that an equalization of pressure between the middle ear and the nasopharynx is possible. The paranasal sinuses should be clear. A roentgenographic study may be necessary; this should always be done with the patient in an upright position to insure visualization of a possible fluid level.

Chest films constitute an important part of the examination. The radiologist must search for (1) broncholiths, (2) blebs, bullae, or pneumatoceles, (3) bronchiectasis, and (4) cavitation. A minimal examination should consist of two posteroanterior films, one of which is made at the usual maximal inspiration and the other at maximal expiration. Diaphragmatic motion is hereby evaluated, and minor degrees of obstructive emphysema can be observed, as in certain cases of asthma, enlarged hilar nodes, and unsuspected tumors. When indicated, the appropriate lateral view and apical lordotic view should be obtained, to visualize the apical region when it is obscured by the overlying first rib and its cartilage.

Technical instruction • If a preliminary physical examination becomes mandatory for membership in a scuba organization, the physician can play a major role in preventing the injuries associated with scuba diving. Items which should be discussed are the signs of vertigo and nitrogen narcosis, proper breathing technics, selection of face masks and compressed air, precautions on length of submersion versus depth, and the absolute necessity for constant exhalation during ascent.

During descent, the most hazardous problem is vertigo. The scuba trainee should be versed in the symptoms of vertigo (such as nystagmus and disorientation) and should be taught that, if vertigo strikes, he should stop the descent, activate flotation devices if available, and move only in the direction of his own exhaust bubbles. He should accept assistance from other divers and learn to rely on them. He should also be taught to go to the aid of another stricken diver, guiding him to the surface and out of the water.

Mild forms of vertigo are frequently experienced during descent because of the head-down position of the swimmer. This orthostatic dizziness is usually mild and transient, and it disappears when the upright posture is resumed. The preventive pharmacology is the same as for barotitis media descendans.

The new enthusiast should familiarize himself with the various face masks available. Face masks which do not provide for air space to nasopharyngeal continuity are positively prohibited. The trainee must be taught proper and relaxed breathing technics.

To avoid intoxication from oxygen and carbon dioxide, the scuba diver must be cautioned never to fill his air bottle with anything but compressed atmosphere purchased from a reputable supplier. Note in the chart that oxygen intoxication may occur at depths in excess of 260 feet even if compressed air is breathed.

The examining physician should caution the trainee to distrust feelings of giddiness or silliness caused by nitrogen narcosis. The diver must be taught to be alert for signs of hebephrenic behavior in others and immediately assist them to a depth at which the symptoms disappear. There are no permanent medical sequelae, and the only danger is that the person will harm himself because his judgement is distorted and his sensibilities are impaired. It is quite probable that some euphoric symptoms occur at depths below 33 feet; this may explain in part the enthusiasm of the "scubaddicted."

Rupture of pulmonary alveoli is to be feared. Members of the St. Louis Underwater Recovery Team constantly phonate "ahh" as they ascend. When it becomes necessary to inhale, the ascent is halted and then resumed with the before-mentioned phonation. This discipline is rigidly adhered to, and members meet all conditions by reflexly entering into this practice. The physician should explain the necessity of this action and emphasize its importance to the trainee.

Another important fact is that nitrogen saturation is cumulative, and the amateur should never indulge beyond the extent of two man-diving hours† per 24-hour period. The professionals have recourse to special air mixtures which exclude nitrogen and negate this hazard.

The chart indicates that there is no decompression time required above 35 feet, regardless of the duration of submersion. From studies conducted, this assumption may be incorrect. In one experiment in continuous submersion for 23 hours in depths of only 8 feet, the divers were forced to terminate the experiment abruptly because of the loss of air source. Twenty hours later, one of the divers exhibited polyarthralgia and abdominal cramping that would be difficult to explain on any basis except decompression. It cannot be proved, but it is highly probable that this man experienced mild "bends." Six weeks later, he suffered a retinal detachment, which is an intriguing coincidence.

Prophylactic therapy: clinical study

Another means of increasing the safety of scuba

†One man-diving hour is not necessarily 60 minutes. It is the amount of time required for one diver to consume 72 cubic feet of air. At very shallow depths, this value will be considerably longer than 60 minutes. At great depth, it will be considerably shorter.

diving open to the physician is prophylaxis. To obtain experience in this phase of scuba medicine, a 3-year study was undertaken to examine the prophylactic measures available.

The study encompassed 400 man-diving hours by male and female scuba enthusiasts, ranging in age from the 'teens to the fifties. The subjects were not in any way screened, and were not aware that they were taking part in a clinical evaluation. Since most of the scuba enthusiasts were familiar with the "ear-clearing" drugs, their use was unanimously accepted. Consequently, it was not too difficult to introduce new compounds to the participants in the study. The drugs used were topical sprays, tablets, and multiple purpose complex tablets. From the results of this study, it was hoped that a formulation for a single tablet would evolve. Nasal decongestants, bronchodilators, antihistaminics, expectorants, and enzymatic anti-inflammatory drugs were given to scuba enthusiasts before their dives. Many compounds were tested, and a group was finally selected.

Results • During the first half of this study, various single drugs and combinations were used. One product offering three desirable pharmacologic effects was selected and used throughout the last half of the study. The decongestant-bronchodilator-antihistaminic tablet selected (Dimetapp Extentabs) contains 15.0 mg. of phenylephrine hydrochloride; 15.0 mg. of phenylpropanolamine hydrochloride, and 12.0 mg. of brompheniramine maleate. These tablets met the criteria of a practical prophylactic drug. Their onset of action was prompt, and their effect was maintained for 10 to 12 hours. The scuba divers were given one tablet 45 minutes prior to entering the water.

Potassium iodide, one of the more commonly known expectorants, proved satisfactory in our studies. Glyceryl guaiacolate (Robitussin) was also tried. Since comparative studies in animals[4,5] indicate that it causes a higher percentage of increase in output of respiratory tract fluids, which are less viscid and contain less mucus, it is probably the expectorant of choice. It is given 45 minutes before entry.

Anti-inflammatory protection is secured from chymotrypsin (Chymoral), 100,000 units taken 45 minutes before entry. Some divers find it helpful to dissolve licorice in their mouths during the period of submersion. This "cough remedy" is available in any drugstore, but may be distasteful to some and objectionable because of its cathartic properties.

Through the prophylactic use of these three drugs, all given 45 minutes prior to diving, injuries were held to a minimum and no serious sequelae resulted.

Two cases of barotitis media ascendans were encountered. One was produced by face mask leakage while the diver was ascending through a plankton layer. No hemorrhagic response was observed in either case, but severe pain was the presenting symptom. Both responded well to catheterization of the eustachian tubes and administration of nasal decongestants. Antibiotics were not given. There were no complications.

Two retinal detachments occurred among the "scubaddicted" not participating in this study. Both of these occurred weeks after diving; in one it occurred 6 weeks after an experiment in continuous submersion for 23 hours. Although this cannot be linked directly with barotrauma, a possible association appears feasible.

No statistics can be offered on the efficacy of bronchodilators since no controls were used. The two divers with chest accidents of ascent, whom I saw, were not protected by prophylactic administration of bronchodilators.

Discussion • It was determined that a practical pre-dive prophylactic agent against barotraumatic injury should meet the following requirements: (1) immediate definite pharmacologic effect; (2) absence of paradoxical response; (3) no nausea or drowsiness; and (4) no rebound phenomena during the diving period (at least 6 hours).

Nasal decongestants shrink the nasopharyngeal mucosal surfaces, allowing the eustachian tubes to be barocanulized with less effort and more certainty. The enzymatic anti-inflammatory agents also promote barocanulizability by helping to decrease the inflammatory response around the internal orifice of the eustachian tubes. Antihistaminics prevent a vasomotor response in the nasopharynx when allergens contact the conjunctivae. These three types of drugs, therefore, aid in the prevention of barotitis media, barosinusitis, vertigo, and subjunctival hemorrhage by keeping the nasal passages, sinuses, and eustachian tubes clear.

It is apparent that maintenance of maximal bronchial caliber lessens the probability of disastrous rupture of pulmonary alveoli. The results of these catastrophes are so horrible to contemplate that any promise of help is readily acceptable to the diver and serves as a psychic support. Air embolism, like shark attack, is difficult to dismiss with mere statistics.

Since scuba air has a marked drying effect, it appears logical that much benefit would be derived from keeping the tracheobronchial secretions as fluid as possible. Practically no expectorants augment the volume of sputum ("expectorates"); but the volume of respiratory tract fluid, which, in nearly all cases, acts as a demulcent to the irritated lining of the respiratory tree, is not expectorated but rather carried up the trachea and then automatically swallowed.[6] The effect of an increase in the respiratory tract fluid caused by expectorants can be further assisted by the use of enzymatic anti-inflammatory drugs that aid in liquifying these secretions.

Summary

The need for the physician to become aware of barotrauma has been substantiated. The types and

mechanisms of barotraumatic injury have been discussed and their treatment reviewed. The pharmacology of prevention utilizing an antihistamine-bronchodilator, nasal decongestant, "expectorants," and an anti-inflammatory agent, and the rationale of their use, have been discussed. An attempt has been made to clarify the role of the physician, and specific therapeutic methods and directions for prophylaxis have been outlined.

Appreciation is expressed to the St. Louis Underwater Recovery Team for its tireless participation; Dr. Don Kinderfather of the Metropolitan YMCA for participation and technical help; Mr. Joseph Jaeger, Jr., of the Missouri State Park Board, for the use of facilities, without which this study could not have been done; and Dr. Raymond Kohler, now deceased, for crystallizing the motivation for this paper.

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Juvenile rheumatoid arthritis (Still's disease)

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The cause of Still's disease, also known as atrophic arthritis, is still unknown. In childhood it is recognized as a slowly progressive, generalized collagen disease which affects the lungs, pericardium, lymphatic tissue, and skin. Polyarthritis with arthritis becomes the most prominent feature. Still's triad, which usually occurs in children of about 5 years, consists of chronic arthritis, generalized adenopathy, and splenomegaly.

Incidence

Although the incidence of this disease is not known, it is not rare. It has been reported that an estimated equal number of new cases of atrophic arthritis, juvenile diabetes, and nephrosis occur each year. At least half of these patients have a familial history of rheumatoid disease. Females are affected twice as often as males, and white persons more often than Negroes. Persons from all social and economic groups may contract the disease. However, a greater susceptibility is shown in patients subjected to fatigue, infection, and emotional strain. The disease is more commonly encountered in temperate climates than in tropical or very cold countries.

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Etiology

It is generally accepted that the cause of atrophic arthritis is unknown. A great deal of disagreement exists among investigators. Some have excluded the possibility of infectious origin, stating that attempts to isolate causative infectious agents from blood, joint fluid, or other potential infected areas have failed. However, there are those[1] who claim the hemolytic streptococci have been cultured most frequently from areas of pathologic change. Hypersensitive response is considered by many to be a definite factor in the etiologic mechanism. However, this has not definitely been proved, nor can it explain the entire pathologic state. In final analysis the only related factors influencing the expression of the disease are heredity, environment, constitutional state of the patient, and psychologic stress.

Clinical manifestations

The onset may be gradual or abrupt. In the acute fulminating case, the symptoms will begin after an

infection in some part of the body, most commonly the respiratory tract. The pattern is that of spiking fever, adenopathy, splenomegaly, anemia, and arthralgias. Arthralgias may be preceded, by a considerable period, by the insidious development of joint stiffness, swelling, pain, and eventual limitation of motion, which may at times be localized for months or even years in a single joint.

An anxious or worried expression is noted in the child with acute illness. Synovial thickening and periarticular swelling are the early joint changes. This is followed by spindling or fusiform changes of the fingers; these are the most common finding in over half the children.

Because of pain at the joint levels, there is limitation of motion. The joints may be warm, but not tender, and the erythema is not extensive. The lower extremities are most frequently involved at the early onset of the disease. Although knee joints may be involved, changes are frequently noted at the cervical spine and temporomandibular joint, which may cause micrognathia. Flexion contractures may result from limitation of motion. This frequently produces residual deformity unrelated to the destruction of the articular tissues.

Temperatures may range from 99 to 107 F. During the acute fevers these patients do not appear nearly as ill as one would suspect from the temperature elevation. This should raise some suspicion as to the diagnostic possibility of juvenile rheumatoid arthritis.

Of considerable importance is the occurrence of a rash that has a significant relationship to the diagnosis of Still's disease. The primary lesions are small salmon-pink macules with pale centers. As a result of coalescence of the macules there frequently will be a blotchy appearance. At the height of the fever, the rash appears evanescent; however, it disappears quickly.

Over 10 per cent of these patients will have some myocardial involvement, with electrocardiographic changes consisting of slight prolongation of the P-R and Q-T intervals with inversion of T_2 and T_3. Murmurs have been described quite frequently. However, these are generally of functional origin, related to the hemic effects of anemia or hyperfebrility.

Changes in joints are as follows: The periarticular tissues are first involved, with edema and some thickening of the capsule of the joint. During this period, the synovial reaction or increase in the amount of joint fluid is highly noticeable. Following this, proliferation of the synovial and joint capsule occurs, with villus formation, growth of granulation tissue, and formation of adhesions and consequent limitation of motion. As the process continues with increased involvement of the joint space, fibrosis and ankylosis of the involved joints occur.

Diagnosis

The roentgenographic findings are quite characteristic in juvenile rheumatoid arthritis. The initial appearance is of soft tissue swelling, with periarticular changes and some evidence of thickening of the synovial membrane. Joint fluid is increased because of inflammation of the synovial cells. Gradual destruction of the cartilage and bone substance occurs in the latter stage of the disease process.

Laboratory studies include many tests, none of which are specific for this disease entity. Sedimentation rate is elevated, and the C-reactive protein is positive during the acute phase. There is usually a mild elevation in the leukocyte count with a reduction in the hemoglobin level. Albuminuria is common during febrile activity. The antistreptolysin-O-titer is seldom elevated; gamma-globulin-coated latex or bentonite particles have been studied, but these are rarely found in children. Tests for lupus erythematosus cells are negative. There may be an increase in the serum globulins, with a reversal of the albumin-globulin ratio, secondary to the stress reaction.

Clinical symptoms are as already described. Exclusion of other disease processes, especially rheumatic fever, aids in making the diagnosis.

Treatment

The main purpose of this paper is to introduce a new application of a combination of medications which proved dramatically effective in the case presented in this report.

Basic therapy is purely symptomatic and of a long-term nature. Because of the unpredictable exacerbations of the disease and the resultant discouraging effects, the evaluation of the therapy is difficult. A team of workers may be required, including the occupational therapist, psychiatrist, orthodontist, and others, with the coordinator being the child's physician. The principle of treatment is to prevent any residual deformities, to maintain the average rate of growth and development at as nearly normal a level as possible, and to attempt to maintain adequate mental and emotional status.

Physical therapy with both active and passive exercises, along with regulation of bed rest during the acute phases, have been of some benefit. However, there are changes in attitude with regard to bed rest even during the acute phases, and opinions vary as to the effects of weight bearing on damage to the joints. There seems to be a feeling that absolute bed rest even during the acute phase is not necessary. Cortisone in a dosage of 4 to 5 mg. per kilogram of body weight per day, or prednisone, 1 mg. per kilogram of body weight per day, is prescribed. Salicylates are satisfactory for alleviating pain and stiffness and reducing temperatures. Maintenance of blood levels of 25 mg. per 100 cc. is recommended, or doses of 100 mg. per kilogram of body weight, divided into four to six doses per day.

This paper reports introduction of a new combination of therapeutic agents: Adenolin (adenosine-5-monophosphate), which is a natural component

Still's triad, which usually occurs in children of about 5 years, consists of chronic arthritis, generalized adenopathy, and splenomegaly. Although the incidence of the disease is not known, it is not rare

of muscular tissue, a mononucleotide yielding on hydrolysis a molecule each of adenine-D-ribase and phosphoric acid, in combination with Medaprin. This combination was first used by a staff member of our hospital, in the case reported below; it provided an excellent response, as the results indicate.

Case report

The first part of this report was obtained from the records of the University of Michigan Hospital in Ann Arbor, where the patient was hospitalized on January 30, 1958.

This boy was in good health until November 1955, when he was 3 years old. At that time, he developed fever and bilateral ankle pain with redness and swelling. These symptoms and signs gradually subsided except for a swelling in the ankle. The child was well until December 1956. At that time, he apparently fell down some stairs and "became paralyzed" from the neck down. This cleared on the following day.

In January 1957, the youngster again developed a high fever and pain in both ankles and knees, and swelling recurred. He was taken to Detroit Children's Hospital where a diagnosis of rheumatic fever was made. At that time he was supposed to have had a heart block. He was discharged on a medication schedule of penicillin and aspirin, and he was to remain at absolute bed rest. The regimen was carried on for 6 months. In September 1957, the child was allowed to go to school for 2 hours a day. At that time his electrocardiogram was normal, but his sedimentation rate remained elevated.

In November 1957, he suddenly developed recurrence of the ankle and knee swellings and swelling of the proximal interphalangeal joints. The areas were so tender that he could not tolerate even the weight of a bed sheet. At that time he also developed exertional dyspnea, fatigue, and anorexia. These symptoms and signs subsided as previously, however. He was left with some swelling in the ankles. Since the onset of that illness in November, his weight dropped from 72 to 56 pounds. Until the time of admission to the University of Michigan Hospital, he had not had steroid therapy.

Neonatal history and other medical history were not contributory. His early growth and development were within normal limits. His immunizations were complete.

On admission to University Hospital in Ann Arbor, his temperature was 99 F, blood pressure was 110/70, pulse rate was 100, and respiration rate was 24. At that time he was a well developed, somewhat thin little boy in no acute distress. Examination of his ears, nose, and throat had essentially negative results. His lungs were clear to percussion and auscultation. The heart rate was 100 per minute, with a sinus rhythm. There was no clinical cardiomegaly. A grade I, soft, systolic murmur was heard at the fourth left interspace just to the left of the sternum. The liver, spleen, and kidneys were not palpable. There was no abdominal tenderness. Examination of the joints revealed swelling of both wrists, ankles, and hips, and the right elbow. There was some fusiform deformity of the proximal interphalangeal joints. There was a 20-degree limitation of extension of the right elbow, and a 10- to 15-degree limitation of extension of the left elbow. Range of motion of the right hip was only minimally restricted, with only mild discomfort on the extremes of range of motion. Examination of the left hip, however, revealed a 30- to 40-degree flexion contracture; it flexed from only 75 to 80 degrees. Adduction was normal at 10 to 20 degrees, but abduction was markedly limited, only 10 to 15 degrees.

Roentgenographic examination revealed osteoporosis of the carpal and metacarpal bones, as well as cystic formations in the subchondral bones of the hips. On an electrocardiogram taken on January 31, 1958, there was a slightly prolonged P-R interval. Review of the electrocardiograms from the previous hospitalization and a repeat electrocardiogram taken at University Hospital on February 25 revealed a prolonged P-R interval and first degree atrioventricular blocking.

Urinalyses during this hospitalization were negative. The patient's hemoglobin on January 31 was 9.3 grams, and at the time of discharge it was 10 grams. Sedimentation rate on January 31 was 12 mm. per hour, but on February 7 it had gone up to 54 mm. per hour and remained in the 50's at the time of discharge. Leukocyte counts were not remarkable during this hospitalization. His serum proteins totaled 9.91; albumin, 3.63; and globulin, 6.28. His blood-urea nitrogen was 13 mg. per 100 cc. C-reactive protein was plus 5. Blood salicylate levels on February 13 were 22 mg. per 100 cc., and on February 27, 18.3 mg. per 100 cc.

This child's hospitalization was characterized by a marked febrile course. He began having fever on February 3, and he was not afebrile until February 24. This was a spiking-type of temperature curve that at times reached 104 F. His weight remained the same during hospitalization, at approximately 53 pounds. Initially, he was placed on a high protein diet, and received 5 grains of aspirin three times a day. This was increased on February 5 to 10 grains

of aspirin three times per day with meals. On February 22, because of failure of response of his fever to aspirin therapy and persistent discomfort in his joints, he was placed on steroid therapy: 2.5 mg. of prednisolone twice a day during his hospitalization. He also was given ferrous gluconate in the hope that his anemia might respond to iron therapy. At the time of discharge, he was given 2 cc. of Imferon intramuscularly for this same purpose.

During his hospitalization the child was seen by the doctors in the Arthritis Research Unit who assisted in the construction of his program. He was also seen in the University of Michigan Orthopedic Department, where assistance was also given in the construction of his medical regimen. He was given special shoes to correct his varus deformity. The Physical Medicine Department treated him with contrast baths and active exercises to maintain motion in the wrists and ankles and to overcome the present observed tendency to knee flexor tightness. This department had instructed the mother in home physical therapy.

At the time of discharge from University Hospital, the patient's program consisted of (1) physical therapy to be carried out at home, and (2) Medrol, 2 mg. twice daily, and aspirin, 0.6 gram three times a day with meals. He was also to take one tablet of multivitamins twice a day. It was recommended that the child participate in all types of activity, with the sole limitation that he avoid becoming excessively fatigued. It was recommended that he return to school. It was hoped that he would be able to live as full a life as possible with his only limitations being those placed by his disease.

This patient was admitted to the Detroit Osteopathic Hospital January 27, 1960, because of severe myalgia, arthralgias, edema, restriction in extension and flexion of both upper and lower extremities, and deformity of the articular processes of both hands, elbows, and knees.

The history indicated that this process had been present for a period of over 4 years. As outlined before, this child had been evaluated at Children's Hospital in Detroit in 1956, at which time a diagnosis of rheumatic heart disease was made. In 1958 the diagnosis of Still's disease or juvenile rheumatoid arthritis had been made at the University of Michigan Hospital.

During the 2 years since his discharge from the Ann Arbor hospital, this child had had intermittent fevers of a rather severe nature, requiring therapy and care. He had been severely ill over the past 2 years with complaints of myalgia, arthralgia, and high fevers. The pain had become so acute that the mother stated that it was difficult even to move him, and just the contact of any clothing or a light sheet would cause a great deal of discomfort. He had been receiving penicillin for 1 year and 3 months. Decadron (dexamethasone) had been administered for the past 18 months. However, despite this management the child's condition had become progressively worse. He had acquired the "steroid face" or "moon face," because of the duration of therapy. Despite this factor, the pain had persisted and become more severe and more frequent, to a constant state, along with frequent temperature elevations.

There are those who claim that hemolytic streptococci have been cultured most frequently from areas of pathologic change. Hypersensitive response is considered by many to be a definite factor in the etiologic mechanism

Therapy over the past 1½ years had consisted of Decadron tablets, two daily (0.75 mg.), Diuril, vitamin therapy, aspirin tablets, one four times daily, penicillin tablets, two twice a day, and a penicillin injection each month.

The remainder of the past personal history was noncontributory. Oddly enough, the family history showed no relation to arthritic disease processes or evidence of chronic collagen diseases. Extensive effort was made to determine any familial contributory factor. However, none was determined.

Physical appearance on admission was that of an 8-year-old white male obese child, exhibiting acute pain to touch in all his extremities. His weight was 94 pounds; he was 51 inches tall; his blood pressure was 118/72. The typical moon face was noted as a result of steroid therapy. There was evidence of both nasal and pharyngeal inflammatory changes of a mild degree. The cervical nodes were palpable, primarily the anterior chain.

Auscultation of the precardium revealed no evidence of cardiac involvement at this time. The apical rate was 108 per minute, of a normal sinus mechanism. There were no murmurs noted, nor was there evidence of cardiomegaly, either to percussion or auscultation. The point of maximum intensity was at the normal position. Auscultation of the pulmonary parenchymal fields had results entirely within normal limits. The aeration was good. There were no adventitious sounds noted.

The abdominal examination showed evidence of splenomegaly; however, the liver was normal, and there was no evidence of tumor formation. Lloyd's, Murphy's and McBurney's signs were absent, indicating normalcy at these points. Neurologic findings were considered to be within normal limits. Deep tendon reflexes were equal and active bilaterally. Funduscopic and cranial nerve surveys were also felt to be negative.

The most interesting findings were those of the extremities, as would be anticipated. The extremities revealed evidence of arthritic changes involving all the joints. There was a marked limitation of

motion to flexion and extension in both upper and lower extremities; this had been present for a considerable period. Joint stiffness, swelling, and pain were quite severe, accounting for the restriction of activity. Spindling fusiform changes in the fingers were present to a rather marked extent. There was only a mild degree of tenderness to touch elicited at the hands, elbows, and knees. There was some mild redness; more significant was the warmth of the areas touched.

Although his temperature was normal on admission, on the third hospital day it rose to 102 F. Intermittent temperature elevations were present throughout the hospital period, to the point of 104 F. on the eighth hospital day. His temperature had wide diurnal fluctuations, ranging anywhere from 100 to 107 F., and elevation continued for prolonged periods. After the sixteenth hospital day, the patient's temperature dropped to 101 F. and remained relatively normal throughout the remainder of the hospital period. It was at this time that a new combination of therapeutic agents was introduced. The youngster remained afebrile for more than 2 weeks before his discharge.

Roentgenographic evaluation was conducted, to re-evaluate the osseous activity. There was evidence of edema involving all the extremities, elbow, wrist, hands, knees, and ankles. There was also evidence of osteoporosis. The articular surfaces, especially the elbows, showed some irregularity with some narrowing of the joint spaces.

Extensive laboratory surveys were conducted. Sedimentation rate was markedly elevated. The electrocardiogram, which is frequently found to be positive in these patients, fortunately showed no evidence of carditis; findings were entirely within normal limits. The C-reactive protein was plus 2. There was a mild degree of anemia. The leukocyte count was quite severely elevated. The antistreptolysin titer was normal. A febrile agglutination series was performed, and results were deemed negative. The nonprotein nitrogen value was elevated for a time; however, prior to discharge this was repeated and was found to be normal. Latex study was done; however, results were not of any significance. Heterophil and Hanger's studies were also negative. The total serum protein and albumin-globulin ratio showed little change at this time.

At this point the diagnosis was made of Still's disease: juvenile rheumatoid arthritis.

The patient was subjected to a new regimen which has proved to have tremendous clinical benefit. A combination of Adenolin, 1½ cc. intramuscularly every other day, and regulated according to the clinical response, along with the utilization of Medaprin tablets, one four times daily, proved to have a dramatic effect in this patient. In addition to this medication, Natrascorb, 250 mg. twice daily, Vi-Aqua syrup, 1 dram daily, Feosol capsules, one daily, and syrup of Achromycin, 1½ drams twice daily, were given for an additional period of 48 hours following the time of discharge.

This patient's response in a period of 2 weeks was quite dramatic. The sedimentation rate and C-reactive protein were normal at the time of discharge. He was afebrile for about 2 weeks before discharge. Most dramatic was the fact that the patient was for the first time in 4 years completely symptom-free, without joint pains or restriction of motion in any extremity. The child was cheerful and had a tremendously improved outlook. The mother exhibited her gratitude and amazement at the results; she had heretofore given up any hope of relief for this youngster. The patient was no longer receiving analgesic medications, penicillin, or Decadron.

At the time of discharge his medication schedule had been reduced to only Adenolin and Medaprin. He has been seen periodically during the past 10 months. He is able to walk and participate in activities at school, and he has become an excellent student. The mother has had very little difficulty with him. He has had only occasional temperature elevations.

Although the prognosis, of course, has been not too encouraging, the fact that the patient has had such a tremendous response to the medications employed may provide a more favorable outlook for these patients.

Summary

This paper has reviewed the etiologic and clinical manifestations of juvenile rheumatoid arthritis. A case was presented illustrating a new combination of medications which had effective results. These medications, Adenolin and Medaprin, may prove to be a more advantageous means of treatment of atrophic arthritis. A new product is currently being introduced on an experimental scale which permits the parenteral administration of this combination of drugs as a single injection. Since the results have been so encouraging in this patient, it is hoped that the crippling effects of this disease process may be delayed or completely averted in others.

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The diagnosis of pulmonary embolism and infarction: I. Clinical and electrocardiographic findings

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Pulmonary embolism and infarction present a difficult diagnostic problem. This is mirrored in the estimation that a large number of cases of pulmonary embolism go undiagnosed and unsuspected. Most physicians are not aware of the high incidence of pulmonary embolism; many are not aware of the varied clinical manifestations that may represent the only evidence of the pulmonary vascular insult. As a result, the incidence of undiagnosed cases remains high.

It is our purpose in the two sections of this paper to correct misinformation; to delineate the clinical, electrocardiographic, and roentgenographic manifestations that may occur; and to present schemes and methods for a more successful approach to the diagnosis of pulmonary embolism and infarction.

For this study, 70 cases of pulmonary embolism were reviewed. They represent the total of diagnosed cases over a 30-month period, from November 1957 to May 1960, in a 230-bed general hospital. Of these 70 cases, 13 were eliminated because of the absence of sufficient evidence to make a relatively certain diagnosis of pulmonary embolism. Therefore, our statistics are derived from findings in 57 patients, of whom at least 33 were attended by one or both of the authors during hospitalization. In 23 cases the diagnosis was made or confirmed at autopsy. In the remainder of the cases, the diagnosis was made clinically, with the aid of electrocardiograms and x-ray studies.

Part II, on roentgenographic findings and differential diagnosis, will appear in the August issue of THE JOURNAL.

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Incidence

The high incidence of pulmonary embolism is stressed by Israel and Goldstein,[1] who report that it is the most common serious pulmonary disorder encountered at the Graduate Hospital in Philadelphia—more frequent than pneumonia and bronchogenic carcinoma. Short[2] has reported similar findings. Hampton and Castleman,[3] at the Massachusetts General Hospital, noted that evidence of pulmonary embolism was found in 9 per cent of routine autopsies over a 10-year period. In a special study group, however, with more careful examination, they noted an incidence of 14 per cent in 400 cases.

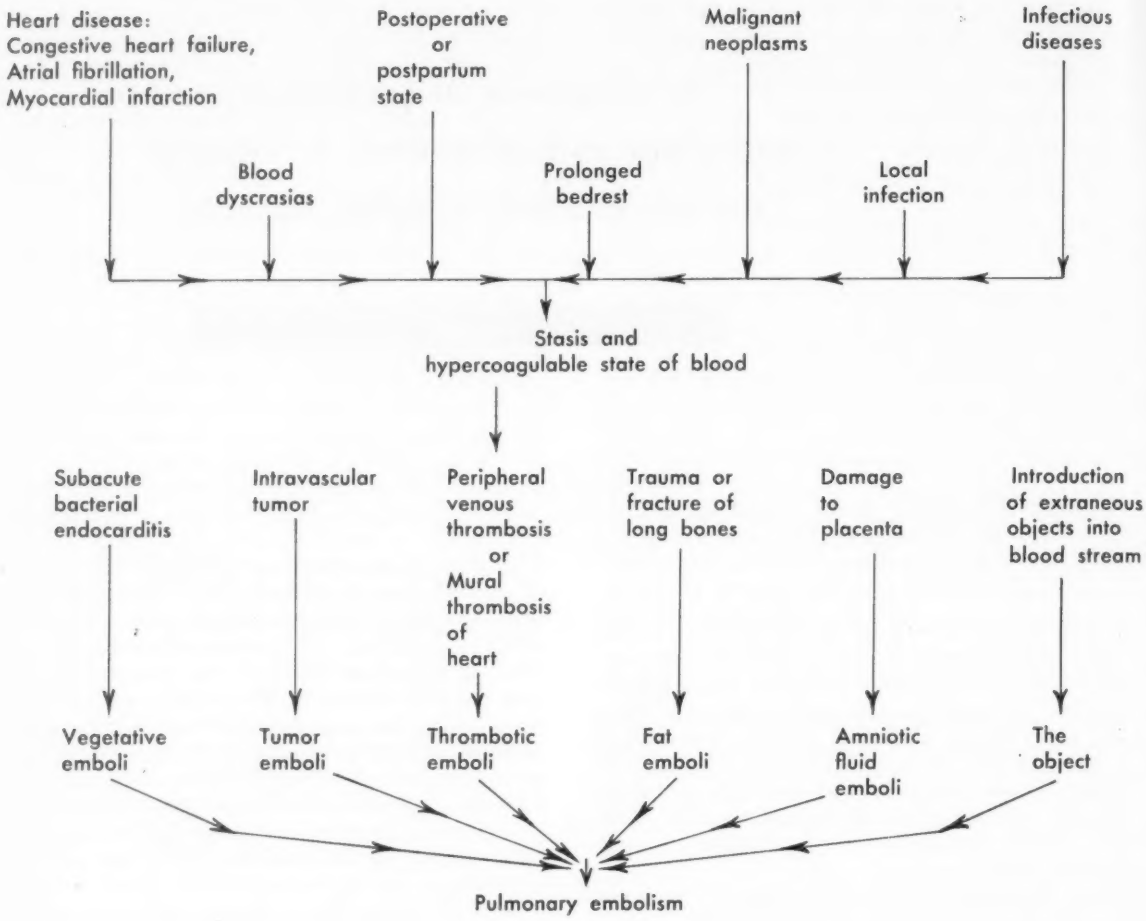
Vital statistics, clinical surveys, and autopsy materials do not give the true incidence of pulmonary embolism as a terminal illness in the general population.[4] The frequency with which pulmonary embolism is detected at necropsy is influenced by the thoroughness and interest of the pathologist; in the routine work of an overtaxed pathologic laboratory, small emboli and infarctions may be overlooked.[1]

The incidence of pulmonary embolism at autopsy is reported in the various general autopsy series as ranging from 5 to 14 per cent.[5] In our series of autopsies, over a 30-month period, there was an incidence of approximately 12 per cent. These statistics are significant in that they indicate that pulmonary embolism should be given a place among the more likely diagnostic possibilities in the differential diagnosis of pulmonary and cardiovascular diseases.

Etiology

It is extremely important to be aware of the etiologic and predisposing factors in the diagnosis of pulmonary embolism. Such awareness may be of prime importance in the recognition of emboli su-

PATHOGENESIS OF THE PULMONARY EMBOLIC EPISODE



perimposed upon cardiovascular and pulmonary diseases, when the ensuing changes might be confused with changes caused by the primary disease process, such as an exacerbation of chest pain after myocardial infarction, due to embolic phenomena. The presence of a common etiologic agent, such as thrombophlebitis, may be important when pulmonary embolism is being differentiated from pneumonia or atelectasis. Also, pulmonary embolism may be the first manifestation of conditions that cause it or predispose to it; occasionally, awareness of these conditions may lead to a retrograde diagnosis of the primary disease process. In 25 of one series of 95 postoperative cases of pulmonary embolism, evidence of the pulmonary accident was present prior to the signs and symptoms of thrombophlebitis.[6]

Age and hospital status • It is noted by Israel and Goldstein[1] that 85 per cent of cases of pulmonary embolism occur in patients over 40 years of age. Our series was consistent with this estimation, in that 88 per cent occurred in patients over age 40. No patients in our series were under 25 years of age, and only 2 were under 30. This is also in general agreement with the ages of incidence of the disease.

It is well established that pulmonary embolism in a general hospital is found more commonly among medical patients than among surgical patients. In the series of Hampton and Castleman,[3] 40 per cent of cases occurred postoperatively, while 60 per cent occurred among medical patients. Our series demonstrated a similar ratio. Twenty-one cases (37 per cent) were in postoperative patients; 33 (58 per cent) were in medical patients; and 3 (5 per cent) were in postoperative patients with medical diseases that would also predispose to pulmonary embolism.

General predisposing factors • Peripheral venous thrombi and mural thrombi of the right side of the heart are the most common primary pathologic entities from which the emboli originate. Vascular insults do occasionally occur as a result of emboli

from other sources. The vegetations on the right heart valves in subacute bacterial endocarditis, or paradoxically on the left side of the heart in the same disease, when associated with congenital heart lesions, constitute important examples of other sources.

Pulmonary embolism is a common complication in subacute bacterial endocarditis; it marked the onset of the disease in 10 per cent of cases in one series.[7] Our series included one patient who presented and was treated for pulmonary embolism on one admission. Antibiotics were included in the treatment because of associated cellulitis in one leg. The patient responded well and was discharged, only to return 6 months later. Attention was then focused upon the mitral configuration of her heart, a marked systolic murmur, a marked fever, and a blood culture which demonstrated Streptococcus viridans. The patient responded well to massive doses of antibiotics.

Pulmonary embolism from the entrance of amniotic fluid into the maternal venous stream during parturition, when fracture or other damage to the placenta occurs, has been described; so has embolism from fat emboli following trauma or fracture of the long bones of the body.[5] Rarely an extraneous object such as a bullet fragment will cause embolism in the lungs.[8] Arendt and Rosenberg[9] report a case of tumor embolism of the lungs from a sarcoma, which characteristically has the capacity to grow into the lumen of a vessel without attachment to the vessel wall.

De Takats[10] reasons that since over 50 per cent of patients manifesting symptoms of pulmonary embolism show no warning in the peripheral circulation, and since a significant number at autopsy demonstrate no source for embolus formation, one may suspect that in at least a small number of cases emboli originate in the pulmonary artery, especially in the presence of pulmonary hypertension.

There are a number of factors which are etiologically related to pulmonary embolism only in that they predispose to or cause thrombophlebitis or mural thrombi, or increase the tendency toward embolism from these sources. The role of these factors in the pathogenesis of the resulting thromboembolic conditions is uncertain. Stasis and hypercoagulability of blood have been incriminated. It is assumed that the factors that predispose to thrombophlebitis and mural thrombi do so by producing one or the other or both of these states.

Heart disease • The factors which favor the development of mural thrombi in the right cardiac chambers, followed by embolism, are those diseases causing stasis within the auricular appendage.[5] The most important of these are congestive heart failure, auricular fibrillation, and myocardial infarction. Obviously, stasis secondary to congestive heart failure includes involvement of the peripheral veins, and the predominance of this condition as a predisposing factor in pulmonary embolism is explained on this basis. Fourteen (24 per cent) of the patients in our series had congestive heart failure as an associated finding. Auricular fibrillation is also felt to increase peripheral stasis.[5] In our series, 6 (10.5 per cent) evidenced this arrhythmia.

Pulmonary embolism is the most frequent of the embolic complications of acute myocardial infarction, according to Friedberg,[11] and is a more common cause of death than is generally recognized. He claims that massive pulmonary embolism accounted for 10 per cent of some 577 deaths in patients with acute coronary occlusion studied by various observers. Embolism may occur from a thrombus on the right side of the ventricular septum or from the wall of the right ventricle, occurring with septal or right ventricular wall infarction, and from infarction of the left ventricle, extending to the right ventricle. Such episodes are usually found during the acute phase of the infarction, before the thrombus has a chance to organize and become firmly attached to the wall of the heart.[12] It should be appreciated that, although infarction may cause thrombi in the right side of the heart, pulmonary emboli most frequently originate in the peripheral vasculature.[11]

It is significant that pulmonary embolism was found in 30 per cent of autopsied patients with cardiac disease by Moran;[13] that Paul, Olwin, and Johnson[12] reported an incidence of 20 per cent of pulmonary embolism in patients having congestive failure, studied at autopsy; that Kinsey and White[14] report an incidence of 48 per cent in the same condition; and that Friedberg[11] relates an incidence of almost 50 per cent in his series of patients with mitral stenosis and right heart failure. We must be cognizant of this high incidence of pulmonary vascular accidents in heart disease, and in myocardial decompensation in particular, if we are to recognize resulting changes in the clinical picture as complications of, rather than manifestations of, the primary disease.

Factors encouraging circulatory stasis • Snapper, Turner, and Moscovitz[15] explain the occurrence of pulmonary infarction in multiple myeloma on the basis of sluggish pulmonary circulation, secondary to high serum globulin, increased blood viscosity, and the tendency of the erythrocytes to formation of rouleaux. We encountered one case of massive pulmonary embolism secondary to multiple myeloma in our series, representing the cause of death. Belt[16] points out that when infection, decreased aeration due to atelectasis, or congestion is present in the lung, pulmonary infarction is likely to occur.

A majority of reports indicate that more than half of the massive pulmonary emboli originate in the legs as venous thrombi.[8] Venous thrombosis occurring after local physical or chemical injury of a vein, and venous thrombosis occurring only in varices, are rarely complicated by pulmonary embolism.[17] The venous thromboses that most frequently give rise to pulmonary embolism include those occurring as complications of postoperative or post-

partum courses, infectious diseases, cardiac disease, blood dyscrasias, progressive malignant neoplasms, or any type of illness requiring prolonged rest in bed.[17] Embolism from thrombosis of the pelvic veins also occurs; it appears most often as a complication of infections or tumors of the bladder, prostate, rectum, uterus, or fallopian tubes.[8]

Postoperative pulmonary embolism is reported as occurring more frequently in the presence of obesity, anemia, varicose veins, previous thrombophlebitis, or any of the predisposing conditions mentioned above. The risk of embolism was noted to be proportional to the severity of the surgical procedure, and was greatest following herniorrhaphy, appendectomy for chronic appendicitis, gastrectomy, cholecystectomy, splenectomy, and hysterectomy.[18] The occurrence of the pulmonary vascular accident is reported to be most common in the second postoperative week.[17] It is estimated that 50 per cent of cases occur then; 25 per cent in the first week; and 25 per cent after the second week.[18]

Our series included 24 cases of postoperative pulmonary embolism. Of these, 16 (66 per cent) occurred in the first week, 4 (17 per cent) in the second week, and 4 (17 per cent) occurred thereafter. There is a significant difference between our series and those reported by others, suggesting that the 25:50:25 percentile ratio cannot be used as a generalization.

Zimmerman, Miller, and Marshall[19] reported an incidence of 36 per cent of carcinoma in 56 patients with pulmonary embolism. Seven patients (12 per cent) in our series had cancer. The significance of these figures lies merely in the probable etiologic relationship of the two diseases, with malignant growth being considered as a predisposing factor to thromboembolic disease.

Increased venous pressure • Embolic phenomena are commonly precipitated by factors which raise the venous pressure and thus dislodge a peripheral venous thrombus.[20] Such factors include straining at the stool, getting in and out of a wheel chair, ambulation after long periods in bed, and exertion. Deep breathing or hyperpnea has been thought to encourage embolism by increasing the rate of venous flow, thereby drawing floating thrombi to the chest.[5]

With these factors in mind, and in spite of increased attention and intensive preventive measures applied to prophylaxis of pulmonary embolism, its incidence has not been lowered, even in institutions where there is special awareness of the problem.[21] However, knowledge of the symptomatology of pumonary embolism, associated with any of these precipitating factors, is suggestive evidence in favor of the diagnosis of pulmonary embolism. Two patients in our series died after sitting on the toilet; whether straining at stool was present was unknown. Whether straining at stool is a precipitating factor, or whether, as will be mentioned later, the urge to defecate represents a symptom of massive embolism, is problematic.

Embolism *vs.* infarction • The difference in meaning between the terms "pulmonary embolism" and "pulmonary infarction" is not generally understood. These terms are not synonymous. Pulmonary embolism means that a foreign object, most commonly a portion of a thrombus, has lodged in a branch of the pulmonary artery, with or without changes in the lung tissue supplied by that branch. When there is a profound change in the lung tissue, which progresses to necrosis, the term "pulmonary infarction" is used.[22] Infarction is not a necessary sequela of pulmonary embolism. Obviously, if death occurs rapidly from the embolism, infarction will not develop. More subtly, however, nonfatal emboli need not produce infarction.

Experimentally, pulmonary embolism in animals and surgical ligation of pulmonary artery branches in humans with normal pulmonary circulation do not produce infarction, as it is defined by the pathologist.[5] The absence of necrosis is explained on the basis of the presence of adequate collateral circulation via the bronchial arterial system.[23] However, if the general circulation of the lung has previously been disturbed, as in cardiac decompensation or chronic disease of the lungs, embolism invites the onset of insidious necrosis of the parenchyma.[5]

Hampton and Castleman[3] noted, however, that in many instances emboli in animals did produce parenchymal lesions which consisted of edema, erythrocytes, and a few leukocytes in the alveoli. In addition, they observed similar hemorrhagic consolidation in the lungs of humans. According to strict criteria, these lesions cannot be called infarcts. They therefore termed them "incomplete infarcts." Such lesions terminate by resolution rather than organization, and are exemplified characteristically in the postpartum or postoperative patient who presents signs and symptoms and roentgenographic changes characteristic of an infarct, but whose signs and symptoms and x-ray changes disappear completely in 2 to 4 days. This same syndrome occurs fairly frequently in the ambulatory patient without indication of a source for an embolus.

The incomplete infarct is also similar to the early stage of a true infarct. It is therefore quite plausible that all these lesions begin in the same way, but that the condition of the lung dictates the course. Thus, an incomplete infarct in the absence of adequate collateral circulation would become complete.[5] Table I, from MacLeod and Grant,[24] shows the pathophysiology of the three possible sequelae of embolism.

Obstruction of the pulmonary artery or its branches may be a result of primary thrombosis rather than thrombosis secondary to embolism. Even on the autopsy table, such differentiation is difficult. Sometimes a small undetectable embolus may initiate a large thrombus.

Criteria have been established to differentiate these two similar pathologic entities. Primary thrombosis is characterized by the presence of evi-

dence of local disease of the pulmonary artery; the thrombus is a cast of the vessel, and there is often lamination of the clot in layers parallel to the arterial wall. When the thrombus is secondary to embolism, no local disease is found in the artery; the embolus is usually coiled; and a source is often found for the embolus.[25]

TABLE I—EMBOLIC OCCLUSION OF PULMONARY ARTERY[24]

Inadequate collateral circulation	Not quite adequate collateral circulation	Adequate collateral circulation
Prolonged ischemia	Anoxia	No anoxia
Destruction of alveolar wall	No destruction	No damage
Influx of blood from dilating bronchial arteries through damaged area	Influx of blood from dilating bronchial arteries confined to vessels with congestion, edema, hemorrhage	No bronchial artery dilation
Complete infarct	Incomplete infarct	Embolization without infarction
Healing by fibrosis	Healing by absorption	—

Clinical findings

The symptoms of pulmonary embolism with or without infarction are protean, and could be caused by several of the common and uncommon respiratory diseases.

Dyspnea • The most common symptom found in our series of cases was dyspnea. Our study suggests that this symptom is universal in pulmonary embolism, occurring at one time or another during the course of the disease. Parker and Smith[5] point out that dyspnea may be mild and evanescent. Surely, many of these cases will be recorded with absence of dyspnea. Three types of dyspnea were noted in our series: typical labored breathing, hyperventilation, and shallow breathing.

The presenting picture of dyspnea in pulmonary embolism varies. It may be mild and of brief duration or severe and persistent. It often begins suddenly and progresses rapidly to gasping respirations. Harris[8] found that the patient can often indicate the exact instant that the respiratory distress began. The breathing, according to Levine,[26] may have a peculiar character, "as if the patient had a foreign body in his throat, a gasping struggle with the mouth wide open, like a fish out of water, but very little air is inspired." The breathing is often typical of that of the underlying respiratory or cardiac disease, such as congestive heart failure or pulmonary emphysema, aggravated by the pulmonary embolus.

Hyperventilation has long been known as a valuable diagnostic sign of pulmonary embolism. It is an important compensatory mechanism, and it may occur despite section of the vagus and sympathetic trunks and despite low carbon dioxide tensions, alkaline pH values, and normal oxygen tensions.[27]

We had one patient in whom hyperventilation was the only presenting symptom for 24 hours, at which time intensification of congestive failure occurred. The patient was resistant to further therapy and died.

Hyperventilation is usually of sufficient magnitude that effective alveolar ventilation is normal or even increased in pulmonary embolism, but occasionally complicating diseases limit the degree of compensatory hyperventilation. Robin and associates[27] reported a case of the "Pickwickian syndrome" (cardiopulmonary failure associated with obesity and hypoventilation) limiting hyperventilation, complicated by pulmonary embolism. We encountered a similar case.

Arterial oxygen saturations were found to be low in the group of cases of pulmonary embolism described by Robin and associates.[27] The degree of unsaturation generally paralleled the degree of occlusion of pulmonary vessels.[21] Three mechanisms for the production of this unsaturation were suggested:

1. Venoarterial shunting
2. Decreased diffusing capacity of lung
3. Relative alveolar hypoventilation.

Regardless of the mechanism, it is obvious that oxygen unsaturation of arterial blood must be at least a contributing cause of dyspnea.

When venoarterial shunting of the lung is the mechanism, the dyspnea of pulmonary embolism will not improve with oxygen therapy. The patient mentioned above, who had hyperventilation, failed to respond to oxygen therapy.

Many patients present a shallow tachypneic type of breathing associated with pleuritic pain. Usually, however, there is more dyspnea than can be explained on the basis of painful limitation of respiratory movements alone.

Pain • Chest pain was the second most common symptom; this was present in 60 per cent of our cases. Its presenting picture is similarly variable, and can be divided into two types: (1) pleuritic pain produced by reaction of the pleura overlying the infarct; and (2) severe anterior chest pain resembling that of myocardial infarction.[22]

Pleuritic pain may be mild and transitory or severe and persistent. It may occur when the patient is still experiencing the initial symptoms of pulmonary embolism, or it may be delayed, occurring 1 or 2 days after other symptoms of embolism have subsided.[8]

When the pain is retrosternal, it may be so similar to that of coronary artery disease that differential diagnosis is difficult. In fact, this pain may be caused by reduction of coronary flow resulting from lowered arterial blood pressure and cardiac output.[28] It may also result from acute distention of the pulmonary artery caused by a mechanical block of pulmonary arterial flow.[8] When such pain is present, however, it rarely has the radiation to the arms that characterizes myocardial infarction.[26]

Cough • This may be present, but it is seldom severe, paroxysmal, or distressing. It results from irritation of the bronchial mucosa, probably within the area of infarction, and from the passage of secretions into normal mucosa. When cough is present, the sputum is usually mucoid and odorless, and it rarely contains pus.[8]

Often, the expectoration consists of almost pure blood, which is usually clotted and rather small in amount. Characteristically, in the hemoptysis of pulmonary embolism, with infarction, the blood becomes darker and scantier as days go on.[29] It is probably associated with the hemorrhage present in necrotic lung parenchyma and from disruption of the bronchial vessels.[5] Massive hemorrhage, blood of fresh appearance or frothy in consistency, or appearing as an effortless welling up in the throat, is not likely of pulmonary embolic origin.[29]

Wright and Foley[22] feel that unless cough with hemoptysis is present, a diagnosis of pulmonary embolism is difficult to make. Parker and Smith[5] relate that hemoptysis has been reported in only 50 to 65 per cent of two series and is therefore less frequent and less important than was originally believed. In our series, a history of cough was reported in only one third of cases, and hemoptysis in only 12 per cent.

Weakness • This is considered a common symptom of pulmonary embolism. It is caused by a reduction of cardiac output resulting from mechanical and reflex blockage of pulmonary circulation.[8] Symptoms characteristic of congestive heart failure dominated the clinical picture in 9 of our patients (15 per cent), with definite evidence of intensification of the disease process in each of these patients. This effect is secondary to decreased cardiac output and increased strain on the right side of the heart.

Abdominal manifestations • Such symptoms of pulmonary embolism are frequently seen. Israel and Goldstein[1] relate that marked muscle guarding simulating true rigidity may occur. They explain this on the basis of referred pain secondary to irritation of the lateral portion of the right diaphragmatic leaf, or from distention of the liver capsule when right heart failure is precipitated. Krause and Silverblatt[30] describe nausea, vomiting, epigastric distress, "butterflies in the stomach," and a sickening feeling in the stomach as occurring in pulmonary embolism. It is our experience that many of these vague mild abdominal symptoms are frequently overlooked or not recorded in the light of other more severe symptomatology.

Neurologic and psychic findings • Neurologic symptoms may occur and may be the major presenting problem. These may be attributed to diminished cardiac output following embolic occlusion of the pulmonary artery. Syncope is a common result. Cerebrovascular insufficiency syndromes, hemiplegia, and convulsive phenomena may occur, but predominantly in elderly patients with advanced cerebral arteriosclerosis.[1] One of our patients, not included in this series, first presented hemiplegic symptomatology.

Neurologic symptomatology may include evidence of the effect on the autonomic nervous system. Krause and Silverblatt[30] report on 10 patients who complained of an overpowering urge to defecate, and who died abruptly thereafter. Of these patients, 8 were autopsied; five showed massive occlusion of a large pulmonary vessel. They discuss 10 patients who were incontinent of urine and feces for variable periods before death. Autopsies were performed on 7 of these patients, and all showed evidence of pulmonary infarction. In our series, 1 patient, with a massive and recurrent embolism found at postmortem examination, died suddenly while sitting on the toilet. Another patient in our series who was found clinically and by roentgenographic evidence to have a small embolism, died suddenly, about 2 months after discharge from the hospital, while sitting similarly on a toilet. As was mentioned in connection with etiology, conceivably this could be a result of "straining at stool," but it would also be consistent with the observations of Krause and Silverblatt.[30] They presume that the urge to defecate occurred with sudden occlusion of a large pulmonary vessel, while incontinence occurred with more gradual occlusion, with adequate time for infarct formation.

Psychic manifestations of pulmonary embolism were reported in 60 per cent of patients in the series of Krause and Silverblatt.[30] These authors described, as nonspecific symptoms that were encountered, restlessness, apprehension, irritability, nervousness, twitching, carphology, combativeness, excitability, argumentativeness, noisiness, confusion, disorientation, depression, concern about themselves, incoherent muttering, desire to die, despondency, unhappiness, crying, drowsiness, lethargy, sinking feeling, and visual hallucinations. Apprehension has been reported by some as a premonitory symptom of pulmonary embolism.[1,22,30] In one case in our series the presenting symptomatology was so predominantly psychic in character that the patient was transferred to a mental institution, only to return with other evidence of pulmonary embolism. It is not clear whether this represents a direct etiologic relationship in this case or an intensification of previous psychiatric disease.

Physical signs • These may be found in the chest when pulmonary infarction occurs secondarily to pulmonary embolism. Such signs occur after one or more of three pathologic changes: consolidation, pleuritis, and elevation of the diaphragm.[2] Accordingly, common findings referable to the chest in our series included restricted chest movements, tachypnea, diminished breath sounds, decreased resonance to percussion, rales, and occasionally a friction rub. These findings are similar to those of Short[2] in England.

Of these findings, those referable to pleuritis are

the most specific. In our series, 7 patients (12 per cent) had evidence of restricted chest movements, 11 (31 per cent) had evidence of a friction rub at one time or another, and 2 (2.5 per cent) had evidence of exquisite tenderness over pleuritic areas.

Israel and Goldstein[1] consider local chest tenderness to be a valuable aid in diagnosis of pulmonary embolism. It may also be elicited in pneumonia with pleuritis, but it occurs far more frequently with pulmonary embolism. It apparently is produced by severe spasm of the intercostal muscles secondary to inflammation of the pleura. In their experience, it usually resulted from emboli with little or no roentgenographic evidence of infarction. When due to pneumonia, the consolidation is usually quite extensive and readily detected by clinical or roentgenographic examination.

Pleural effusion may occur secondarily to the pleuritis. Occasionally it may be of considerable magnitude. When small, physical signs may be minimal or absent. The fluid may appear straw-colored or grossly bloody. Numerous erythrocytes and approximately equal numbers of granulocytes and lymphocytes are found. The effusion may continue for several weeks and lead to considerable concern over the possibility of tuberculosis or neoplasm.[8]

Cardiac and circulatory findings • Precipitation or intensification of heart failure, as mentioned before, occurred in 15 per cent of our series with appropriate signs. Israel and Goldstein[1] report an incidence of 25 per cent in their series. Should acute dilatation of the right heart occur, prominent pulsation along the right sternal border may be evident, together with a loud second pulmonic sound, a pulmonic systolic murmur, and a diastolic gallop rhythm.[5] Cyanosis is not a very common manifestation of pulmonary embolism. It occurs in massive embolic phenomena and when pulmonary edema accompanies pulmonary embolism. It occurred in 7.7 per cent of Israel and Goldstein's series,[1] and in 5.5 per cent of our series.

Hypotension is a terminal manifestation of massive and recurrent pulmonary embolism, but it may occur as a presenting sign. Katz and Saphir[31] describe well-developed baroceptor nerves in the pulmonary artery analogous to those in the carotid sinus mediating depressor reflexes whenever acute pulmonary hypertension develops. They describe a nerve plexus rich in ganglion cells and chromaffin cells, between the pulmonary artery and the ascending aorta, with afferent fibers in the vagi and sympathetics. They consider the possibility that these might mediate impulses responsible for hypotensive reactions. Jesser and de Takats[32,33] present evidence that, with partial occlusion of the pulmonary artery, reflex effects on the heart, bronchi, gastrointestinal tract, and unobstructed pulmonary arteries occur.

Signs of thrombophlebitis may be present, which are strong evidence in favor of pulmonary embolism when respiratory signs and symptoms compatible with this vascular accident are present. Sometimes the thrombophlebitic signs may not appear until about a week after the pulmonary vascular accident. As previously mentioned, the phlebitic source of embolism may never be located. Signs of thrombophlebitis were found in 8 (14 per cent) in our series.

Tachycardia is a very common manifestation of pulmonary embolism. It occurred in 42 per cent in our series, and in 59 per cent of Israel and Goldstein's series.[1] It may be secondary to fever, thrombophlebitis, myocardial arrythmias, or congestive heart failure. Tachycardia and tachypnea together, out of proportion to the patient's fever and pulmonary congestion, should always suggest pulmonary embolism.[1] Frequently it will be observed that the pulse and temperature had been rising for a few days before the more dramatic acute manifestation of pulmonary embolism took place. Levine[26] considers these premonitory features as most likely caused by the underlying thrombophlebitis.

Fever and jaundice • Fever is a common manifestation of pulmonary embolism. It will depend on the degree of infarction or secondary pneumonitis or abscess, and the presence or absence of thrombophlebitis at the site of origin. Some fever is commonly present, and it may be marked.[22] Oral temperatures over 100 F. were recorded in 17 patients (33 per cent) during the course of their hospitalization for this illness in our series. The fever commonly comes on abruptly and usually persists for 5 to 7 days. Unexplained fever in a bedfast patient should suggest the possibility of pulmonary infarction. Since the fever in this condition is a response to tissue necrosis (if not complicated), antibiotic therapy is ineffective.[5]

In Israel and Goldstein's series,[1] clinical jaundice was encountered twice and subclinical jaundice once. All three patients had repeated large pulmonary emboli and right heart failure. It has been suggested by others that the hyperbilirubinemia associated with pulmonary embolism is caused by lysis of the infarct with hemolysis. But in all of these three patients, the direct reacting fraction of bilirubin was significantly elevated. One patient demonstrated evidence of hepatic dysfunction as determined by liver function studies. It is therefore considered that hepatic dysfunction may be the major factor when icterus occurs following pulmonary embolism. We encountered no evidence of hyperbilirubinemia in our series, but serum bilirubin was determined in only a small minority of our cases.

Electrocardiographic findings

The importance of the electrocardiogram as an aid in the diagnosis of pulmonary embolism deserves much emphasis. It is of extreme importance for differentiation when the signs and symptoms of the embolic episode resemble those of myocardial infarction. It may be of singular importance when the signs and symptoms are atypical or masked by

underlying disease, and when they could be explained on the basis of a disease process already known to be present. Furthermore, it presents additional supportive evidence in the diagnosis of cases with more characteristic signs, symptoms, and x-ray findings. As found by Krause and Silverblatt,[30] however, in a 5-year study at Montefiore Hospital, electrocardiograms are usually not obtained early enough, not often enough, and not in serial fashion, when the diagnosis of pulmonary embolism is suspected. They found a pathognomonic pattern in 37 per cent of 62 patients; nonspecific evidence in 23 per cent; and absence of evidence in 40 per cent. Those not having electrocardiographic evidence of embolism were considered as not having had studies taken early enough or often enough.

In our series of 57 cases, 21 (37 per cent) had no electrocardiogram taken at or sufficiently near the time of the embolic phenomenon. Of the 36 remaining cases, 16 (45 per cent) presented patterns which could be interpreted as strong evidence of pulmonary embolism. Twelve (33 per cent) presented evidence supporting the diagnosis.

As related by Wolff,[34] the usefulness of the electrocardiogram as a diagnostic aid in pulmonary embolism may be limited by the presence of changes secondary to pre-existent heart disease, such as acute myocardial infarction. In our series, 8 (22 per cent) demonstrated no electrocardiographic evidence of pulmonary embolism. In 3 of these, changes that could be attributed to pulmonary embolism were obscured by changes characteristic of heart disease.

In Israel and Goldstein's series,[1] "significant changes" were noted in the electrocardiographic pattern in 75 per cent of cases diagnosed as having pulmonary embolism. They define significant changes as transient and characteristic abnormalities whose demonstration in a patient with an appropriate clinical picture supports the diagnosis of embolism. Our statistics compare favorably, in that 78 per cent of our cases with electrocardiograms taken at proper time intervals demonstrated "significant changes."

In the light of our experience and of statistics accumulated from various investigators, electrocardiographic patterns which are encountered in pulmonary embolic episodes can be classified into eight categories, including: (1) complete patterns of acute cor pulmonale, (2) incomplete patterns of acute cor pulmonale, (3) chronic cor pulmonale patterns, (4) elevated S-T segments over right precordial window leads, (5) depressed S-T segments in association with complete or incomplete cor pulmonale patterns, (6) inverted T waves over the right precordium, (7) transient arrhythmias (alone or in association with other findings), and (8) transient right bundle-branch block.

The incidence of these patterns in our 36 cases with significant electrocardiographic changes is depicted in Table II. Although right bundle-branch block and transient atrial fibrillation were not encountered in our series, Israel and Goldstein[1] report each as occurring in about 10 per cent of their series, and other investigators report them as occurring in pulmonary embolism. They were therefore included in our classification.

TABLE II—ELECTROCARDIOGRAPHIC PATTERNS IN PULMONARY EMBOLISM: INCIDENCE IN 36 CASES WITH SIGNIFICANT ECG CHANGES

Electrocardiographic pattern	No. cases
Acute cor pulmonale, full pattern	4 (11%)
Acute cor pulmonale, incomplete pattern	18 (50%)
Chronic cor pulmonale	1 (3%)
Elevated S-T segments over precordial window leads	2 (5.5%)
Depressed S-T segments in left precordial leads	4 (11%)
Inverted T waves over right precordium	2 (5.5%)
Transient arrhythmias (frequently accompanying other findings):	
(a) Sinus tachycardia	8 (22%)
(b) Transient atrial fibrillation	0 (0%)
(c) Ventricular extra systole	2 (5.5%)
(d) Others	0 (0%)
Transient right bundle-branch block	0 (0%)

Cor pulmonale pattern • It is obvious from our study, and it follows in other studies, that complete or incomplete patterns of acute cor pulmonale are the most common electrocardiographic manifestations of pulmonary embolism. There are many isolated electrocardiographic changes that may occur secondarily to cor pulmonale, and they are seldom all present. All varieties of combinations may occur.

Before the advent of the unipolar leads, the McGinn-White pattern was described in the standard limb leads as occurring characteristically in acute cor pulmonale. This included a deep S_1 and Q_3, a "staircase ascent" of the S-T segments in leads I, II, and III, and a positive T_1.[5] The so-called staircase ascent consists of depressed S-T segments in leads I and II, with an elevated or isoelectric S-T segment in lead III. However, only isolated aspects of this pattern may be present in a given case, such as deep Q_3 and negative Q_3. Furthermore, similar evidence may be present in the unipolar limb leads. A deep S wave may occur in a V_L and a deep Q or a small q in a V_F.

Kuo and Vander Veer[35] explain the mechanism of production of some of the findings in the limb leads. With pulmonary embolism, large or small, there is a marked elevation of pressure in the pulmonary artery and in the right ventricle, with resulting dilatation of the right cardiac chambers and with a decrease in cardiac output. This dilated ventricle bulges and projects itself into the anterior plane of the body. In such a position, the electrical potentials of the left arm may develop an S wave, especially in aVL. S waves are present in only a small number of patients with pulmonary embolism, because of the negativity of the right arm potential which goes into the making of lead I. The lowering of the T waves in aVL and L_1 may be related to right ventricular ischemia resulting from increased

intraventricular pressure and diminished coronary flow.

It is explained by Levine[26] that the small r and deep S at lead aVL are recorded at leads III and aVF as a small q and a tall R. At the same time the left ventricular potentials (qR) are transmitted to the left leg and are recorded in leads III and aVF, contributing further to development of Q waves in these leads. Sodi-Pallares[36] attributes this finding of more or less deep Q waves in aVF to extreme clockwise rotation with forward displacement. He relates that the epicardial surface of the right ventricle near the pulmonary conus may give rise to qRS or qR complexes after ligation of the pulmonary artery. Under such conditions, deep Q waves are recorded at the epicardial surface of the right auricle. The dilatation of the right auricle and the positional changes mentioned above favor the presence of Q waves in aVF, and therefore in lead III.

Levine[26] feels that the depression of S-T segments in the limb leads is probably related to subendocardial ischemia. Sodi-Pallares[36] explains positive S-T displacement in aVF on the basis of injured areas which may occur either on the right septal surface or in the free wall of the right ventricle.

A prominent R or R′ may occur in aVR[37] on the basis of the positional changes described above.

Many of the changes in the precordial leads are the result of the same or similar changes in position of the heart. Obviously clockwise rotation as registered in the precordial leads follows the explanation for the standard lead changes. Shift of the transitional zone to the left may result from chance variations in the heart's position, resulting from its normal mobility. However, Levine[26] points out that conspicuous migration of this zone toward the left axilla, if not otherwise explainable, is probably related to clockwise rotation of the heart. Clockwise rotation is usually associated with relative verticalization of the heart, and this might be recorded on the electrocardiogram.[35] Positive S-T segment displacements may occur in chest leads I and II on the same basis as offered for explanation on this elevation in aVF.[1] The qR or Qr patterns found in aVF leads are also found in these two leads. Again, any of these precordial changes may occur alone or in combination with any other change, precordial or standard.

Table III demonstrates the rate of occurrence of the various findings attributed to cor pulmonale in the electrocardiograms of the 36 patients in our series with significant changes.

Many are under the impression that only massive or multiple pulmonary embolism will produce changes in the electrocardiogram. A number of clinical and experimental studies[35] have shown that even with a small embolism there is marked elevation of pressure in the pulmonary artery and right ventricle, and the same changes occur as with large emboli. Phillips and Levine[37] point out that reflex

TABLE III—INCIDENCE OF ELECTROCARDIOGRAPHIC FINDINGS REFERABLE TO COR PULMONALE IN 36 CASES WITH SIGNIFICANT ELECTROCARDIOGRAPHIC CHANGES

	No. patients
Standard leads	
S_1Q_3 pattern	2 (5.5%)
Q_3	3 (8.5%)
S_1 alone	1 (3%)
R in aVR	7 (21%)
Staircase ascent	6 (17%)
Precordial leads	
Clockwise rotation	9 (25%)
Verticalization	1 (3%)
Shift of transitional zone to left	3 (8.5%)

spasm of the pulmonary tree can be induced by small emboli producing pulmonary hypertension. Arendt and Rosenberg[9] postulate the presence of vagal reflexes causing widespread constriction of the pulmonary arterioles, stimulation of the respiratory center, and diminished coronary flow, all of which are additive to the mechanical effect of the embolus.

Multiple and recurrent pulmonary emboli lead to the development of chronic cor pulmonale.[9,38] The electrocardiographic pattern of this condition will not be discussed here.

The remaining patterns found in pulmonary embolism are less common as isolated findings, and are sometimes found in association with each other or with changes characteristic of acute cor pulmonale.

S-T segment and other variations • "Window leads" are those leads in which an R wave is absent, perhaps as a result of previous myocardial infarction, or normally as a result of position of the electrode, which therefore transmits intracavitary potentials. S-T segment elevations have been described in these leads in association with angina pectoris, coronary insufficiency, and positive exercise tests. Pulmonary embolism may produce this same effect by producing coronary insufficiency and subendocardial ischemia.[1]

Sometimes the only manifestations of pulmonary embolism on the electrocardiographic tracing are depressed S-T segments or inverted T waves over the left precordium, evidencing coronary insufficiency.[36] Again, without positional changes characteristic of cor pulmonale, it is difficult to attribute this to pulmonary embolism.

Inversion of the T waves over the right precordium is probably related to a change in the repolarization process in the right ventricle (so-called right ventricular strain or ischemia).[2] The transient inversion of these T waves may be mistakenly considered as diagnostic of anteroseptal infarction. Again positional changes in association are helpful in differentiation.

Transient right bundle-branch block may occur

as a result of the marked rise in right ventricular pressure with its effect on the right side of the interventricular septum, interfering with the conduction through the bundle of His.[39] This may also occur as a transient feature in myocardial infarction,[26] and is therefore more helpful diagnostically if associated with other signs of acute cor pulmonale.

Differential findings • It is obvious from the material presented that the patterns occurring in the electrocardiograms taken in patients with pulmonary infarction are frequently not clear-cut, and that a good knowledge of differential diagnosis in electrocardiography may be necessary to differentiate the tracing from that of a myocardial infarction. The presence of Q waves in leads III and aVF are prime examples of the findings that can be equivocal. Levine[26] points out that it has been claimed that in acute cor pulmonale a prominent Q_3 is not associated with a significant Q in aVF; theoretically this is sound, but in actual practice it is not reliable. Wolff[34] considers duration of the Q wave as the most decisive differential feature: 0.03 second or more for posterior myocardial infarction; less than 0.03 second for positional changes due to pulmonary embolism. The S-T segment elevation in lead III and depressed T_3, which may occur, may add to the confusion. In pulmonary embolism the S-T elevation is usually minimal and is not convex upwards as in myocardial infarction.

Some differential points include the "staircase effect" of the S-T segment in pulmonary embolism; the fact that in pulmonary embolism lead II resembles lead I, while in posterior myocardial infarction lead II resembles lead III; and that p waves occasionally become tall and peaked in leads II and III in embolism.[31] Terminal R waves in aVR and R waves in aVF are more frequently seen in posterior myocardial infarction.[34]

The most important differential points include the associated presence of inverted T waves over the right ventricle, in contrast to the tall upright T waves present in posterior myocardial infarction;[26] associated positional changes characteristic of acute cor pulmonale; and the transitory nature of the electrocardiographic changes as evidenced on serial tracings.[34,40-42]

Occasionally, the electrocardiogram will present a picture which must be differentiated from anterior myocardial infarction; for example, positive S-T displacement in V_1 and V_2, along with wave depression in these leads, and qR or QR ventricular complexes. In a majority of cases of acute cor pulmonale with these findings, they do not extend beyond V_3, which is unusual in anteroseptal infarction.[36] In acute cor pulmonale there may be evidence of subendocardial injury, characterized by positive S-T displacement in a V_R lead; again the most important differential point is the transitory nature of the findings in pulmonary embolism.

From our clinical studies and investigation of the literature it can be surmised that the electrocardiogram can be an important diagnostic aid in pulmonary embolism; that the positional changes secondary to cor pulmonale are the most common electrocardiographic findings; and that serial electrocardiograms must be taken at opportune moments to obtain satisfactory evidence.

TABLE IV—DIFFERENTIAL FINDINGS ON THE ELECTROCARDIOGRAM

Pulmonary embolism	Posterior myocardial infarction
Q_3VF width less than 0.03 sec.	Q_3VF width 0.03 sec. or more
S-T segments minimally elevated in aVF	S-T segments convex upwards
Lead II resembles lead I	Lead II resembles lead III
Staircase effect sometimes present	No staircase effect
Terminal R waves in aVR may occur; S waves in aVL	Terminal R waves in aVL may occur; S waves in aVR
Inverted R waves in right precordium	Tall peaked T waves in right precordium
Positional changes secondary to cor pulmonale	No positional changes
Rapid transitory changes	More gradual transitory changes

Wolff[34] emphasizes the suddenness with which the changes on the tracings occur in pulmonary embolism—in some cases coincident with clinical episodes and in others after a lapse of days or hours. Their sudden disappearance is equally striking; it usually occurs within a week, or days, or hours, but rarely are they present for a longer period.[42]

We therefore recommend that: (1) electrocardiograms be taken as soon as possible after suspected embolic accident; (2) that they be repeated at intervals of hours or days, according to the findings and clinical changes; and (3) that serial tracings be taken over a period of 2 or 3 weeks when initial positive findings occur, if they are not completely absent in a shorter period.

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Preparation and premedication for the first stage of labor

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Labor consists of a series of processes by which the mature or almost mature products of conception are expelled from the mother's body. The first stage of labor is only one phase of this continuous process; it begins with the first regular uterine contraction and ends with the complete dilatation of the cervix.

During the early part of the first stage of labor the uterine contractions are short, slight in intensity, separated by long intervals, and usually do not cause any degree of discomfort. As labor progresses the uterine contractions occur at shortening intervals, increase in intensity, are of longer duration, and cause greater degrees of pain. Indeed, the pains which precede and accompany full cervical dilatation are often of excruciating severity.[1] The pains that the parturient experiences during the first stage of labor are primarily caused by the dilatation of the cervix and lower uterine segment, and to a lesser extent from the contractions of the body of the uterus.[2]

The average duration of the first stage of labor in the primigravida is 12 hours; in the multigravida, 7 hours.[3] Certainly the averages are quite variable, just as the amount of pain experienced by the patient is variable. It should be remembered, however, that the pain during labor is a psychologic interpretation of a physiologic entity and therefore is real.

Javert and Hardy[4] made a fundamental contribution in the recognition of pain during labor by standardizing the character of pain in unmedicated women throughout the various stages of labor. They found that the mean intensity of the first quarter of the first stage was no more than 2 dols,

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and that a majority of the patients considered this to be quite mild. However, the pain progressively increased in intensity, ranging from 3 to 5 dols in the second quarter of the first stage of labor, 5 to 7 dols in the third quarter, and from 7 to 10 dols during the final quarter and through the second stage. Most patients considered the final quarters of the first stage and the second stage to be the most intense pain in their experience. From these studies it can readily be seen that the use of major narcosis is seldom necessary during the initial portion of labor, but we must be cognizant of the need for analgesia during the later phases.

Unfortunately, the relief of pain during labor presents special problems. It is difficult to safely premedicate the parturient without giving some thought to these peculiarities. Probably the best way to illustrate some of these problems related to obstetric analgesia is by comparison with the surgical patient.

In the parturient there are two patients to consider, whereas in the surgical case there is only one. The second patient, the fetus, is known to be extremely vulnerable to sedative and anesthetic drugs. Since these agents readily pass the placental barrier the possibility of inhibiting fetal respiration is always present. This fetal factor, coupled with the probability that the premedication must extend over a period of many hours before delivery, presents one of the most difficult problems in obstetric pain relief.

The inhibitive effect of most analgesic agents on the uterine contractions is another intangible which must be appreciated in the obstetric case. Indeed, the premature and injudicious use of analgesic medication looms large as a cause of uterine inertia and prolonged labor. It should be a firm rule that such medication never be administered until positive proof exists that the cervix is showing progressive effacement and dilatation characteristic of labor in the acceleration phase. Thus, there must be a careful blend of pain relief compatible with progress in labor.

Obstetric patients, unlike surgical patients for planned operations, frequently present themselves near delivery, in precipitous labor, after having eaten a full meal. The possibility of aspiration of vomitus must certainly always be considered when planning premedication for the parturient.

Although many advances have been made in obstetric analgesia and anesthesia, the contributions in this field have not been commensurate with the progress in surgical anesthesia.[5] This is especially true as regards the administrator. To further improve the relief of pain during labor, the anesthesiologist must participate as a member of the obstetric team in a similar manner to his participation on the surgical team. In modern obstetrics there needs to be a closer correlation of the principles of both specialties.

These are some of the factors to consider when premedicating the obstetric patient during the first stage of labor. Since most women in the United States, regardless of propaganda to the contrary, desire and require analgesia and anesthesia during labor and delivery,[6] we should be familiar with the special requirements. As Carroll and Moir[7] have stated, "It is no triumph of the obstetric art to deliver a woman safely but to leave her mind so full of vivid and unhappy memories that she refuses to have another baby."

The preparation and premedication of the obstetric patient actually begins with her first prenatal visit. A strong patient-physician rapport should be established early and maintained through careful antepartum care and frequent discussions. We should educate and reassure the patient concerning the physical facts of pregnancy and the folklore about childbirth. The patient should be told that anxiety during pregnancy is not abnormal, and that childbirth without fear or pain probably never or rarely occurs. This is quite important as many parturients are ashamed of exhibiting anxiety and, after all, the emotional reaction in optimal amounts acts as protection and a means of survival. A good physician-patient relationship is an almost indispensable basic sedative. We have all seen the healthy, carefree, unafraid patient who has complete confidence in her physician progress through the major part of the first stage of labor with minimal discomfort.

Patients who have been carefully prepared for the experiences of labor and delivery will also demand certain preparations on the part of the hospital. The hospital maternity division should be pleasant and comfortable. A study by Hingson and Hellman[6] revealed that more comfortable and pleasant environment, embodying air conditioning, interior decorating, lighting, and therapeutic music, often was as important as doses of the narcotic. Aside from the physical aspects of the environment, each hospital should provide means for complete and safe anesthesia of both the encephalic and anatomic variety. This should hold true for spontaneous delivery, operative delivery, and cesarean section.

There are many varied technics of premedication utilized for the patient in labor, and each has its staunch proponents. Unfortunately, there is no best method or agent; all have advantages and disadvantages. Nor is there a "best variety" so far as fetal outlook is concerned. By this is meant that properly chosen and administered analgesics and anesthetics offer about equal chances of success. Probably the most important single factor in determining the type of premedication utilized in a given case is the degree of training of the obstetrician and anesthesiologist.

It is not within the scope of this presentation to describe each agent and technic available today. Rather, I would stress certain fundamentals peculiar to the premedication of the parturient and outline a method which has proved successful for me in the average normal labor.

There exists a wide range of opinion as to what constitutes adequate pain relief during labor and

delivery. There are those individuals who feel that if amnesia for the experience is obtained, this is sufficient, even if the patient is unruly, hard to manage, and noisy. Others are completely satisfied if minimal relief of discomfort is established, and some still feel that since analgesic agents may be harmful none should ever be given.

In my opinion, the patient should be premedicated in such a manner that when the latter phases of the first stage of labor are reached and delivery is imminent, an anesthesia can be given that will provide for a painless, quiet, orderly procedure. Certainly where possible the level of pain relief should be such that any type of situation may be cared for in a painless manner without the patient screaming and kicking and without the necessity for physical restraint. The days of noisy maternity wards should be past. The judicious administration of analgesia is beneficial to the mother,[8] and the adequate relief of pain will permit more meticulous, more gentle, and frequently easier deliveries, resulting in healthier mothers and more living babies.

We must also be aware of the wishes of the woman who neither wants nor needs analgesia. Obstetricians and anesthesiologists must understand psychoanalgesia and carry it as far as is expedient without prejudice. However, we should always have at hand the analgesia so often required unexpectedly.

When considering the average, normal obstetric patient, the premedication problems are not particularly great if we keep in mind our basic principles. After I have completed my initial examination of the parturient and find that she is in the early phases of first stage of labor, she is admitted to the labor room. Here the usual procedures of cleansing the lower bowel, preparing the perineal region, and obtaining specimens for laboratory studies are carried out. After these preliminaries are completed, the patient is again visited. If labor has not progressed past the early phases and the patient is not particularly uncomfortable, she is assured that normal progress is being made and that I shall be nearby.

As a basic sedative at this time 50 mg. of promethazine hydrochloride are administered intramuscularly. This particular drug has been chosen because of its quieting and antiemetic effect on the patient without causing any major effect on the fetal respiratory system and without maternal side reactions. In a recent study[9] promethazine was evaluated in approximately 5,000 obstetric and surgical patients as a premedicant, antiemetic, and sedative. Of 600 obstetric patients, premedication with promethazine was satisfactory in 82 per cent, as evidenced by quiet, restful sleep and effective analgesia. Incidence of nausea and vomiting in all stages of labor was 3.6 per cent. Hypotension and tachycardia in all stages were notably absent. No untoward effects on the infants were noted. It is thought that if promethazine does cross the placental barrier, it does so with difficulty and slowly in contrast to the barbiturates. Other studies, including those of Burn,[10] Dawes,[11] and Light,[12] have shown promethazine to be an excellent drug for premedication. My experience has proved promethazine to be predictable and effective.

As labor progresses and pain increases, I administer another 50 mg. of promethazine intramuscularly and 40 mg. of alphaprodine hydrochloride subcutaneously. Alphaprodine hydrochloride is used in preference to the other opiates or synthetic opiates because of its rapidity of onset, relatively short duration of maximum effect, and its minimal depression of respiration in the infant. Emich[13] administered alphaprodine hydrochloride in 40-mg. doses to 1,000 women during labor and found that satisfactory analgesia was obtained in 92 per cent. In his series respiratory depression was observed in only 0.9 per cent of the infants. Kane,[14] in a series of 1,000 obstetric cases, found satisfactory analgesia in 98.1 per cent, with no maternal side effects except for 8 patients who complained of dizziness. Fetal respiratory depression was found in 4.9 per cent of the infants, with none being severe or fatal. Alphaprodine hydrochloride, administered in 40-mg. doses with promethazine, has proved very effective in relieving the pains of labor during the major part of the first stage without any untoward effect on the mother and with a very minimal degree of infant respiratory depression. The drug must be administered subcutaneously rather than intramuscularly, however, in order to have this effective and predictable action.

The amount of medication thus far administered will provide adequate analgesia for the average, healthy, well prepared patient to a point in the first stage where a regional block anesthesia can be safely started. If by chance labor does not progress quite this rapidly and more analgesia is necessary, the alphaprodine hydrochloride in 40-mg. doses is repeated every 2 or 3 hours. This amount of medication has not resulted in any major degree of infant respiratory depression or general maternal complication, even if an inhalation anesthetic was given for delivery rather than the regional block. Many authorities recommend alphaprodine in 60-mg. doses; however, it has been my experience that the larger dose was no more effective in relieving pain. Occasionally if a third or fourth injection of alphaprodine is necessary before the patient is ready for her anesthetic, levallorphan tartrate, 1 to 2 mg., is given with the last dose of premedication. A premixed solution containing alphaprodine hydrochloride and levallorphan in a 50:1 ratio is available; however, I have not found it necessary to administer levallorphan with each dose of the analgesic.

When the patient is admitted in the latter phases of the first stage of labor and is quite uncomfortable, the promethazine and alphaprodine combination is given immediately and then the patient is prepared for the labor room. If the patient is far enough advanced in the first stage, only the promethazine is given and then a regional block is started.

Even though scopolamine is often an excellent drug to include in the premedication routine, I have not used it in my usual technic because of the marked excitability that it produces in certain individuals. Also, I do not desire my patients to have amnesia for the experience of labor and delivery.

Summary

There are many varied agents and methods of premedication of the obstetric patient, and it is not within the sphere of this paper to examine the merits of each; however, certain general principles have been discussed, and a method of premedication has been presented. Certainly this must be altered at times, but with the well prepared patient, where physician-patient rapport is well established, the agents and method presented have proved very satisfactory.

For the best appreciation of pain relief in labor and delivery, one needs constant revision and analysis of data with a fair and open mind, comparing the new with the old in formulating opinions.

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Pulmonary roentgen changes following cholecystectomy and their relationship to respiratory morbidity

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The initial stimulus for this investigation was provided by a difference of opinion among members of the Department of Radiology at Metropolitan Hospital regarding an oblique linear density seen on a posteroanterior chest film obtained 4 days following a cholecystectomy on a 43-year-old Negress, who presented a nonspecific clinical picture of moderate respiratory distress. The usual roentgen differential diagnostic possibilities were discussed. A superficial investigation of the available references failed to provide any information on the normal appearance of the chest following abdominal operations. Thus the difference of opinion remained unresolved.

It was decided at that time not only to follow up on the particular case in question but to obtain posteroanterior chest films on all patients undergoing cholecystectomy, at arbitrarily chosen postoperative intervals of 3 and 7 days. Pulmonary roentgen changes were demonstrated in 5 of the first 10 cases; so the study was continued until 70 cases had been collected. An intensive review of the literature was undertaken at the same time. The relative paucity of information regarding the normal and abnormal roentgenographic appearance of the chest following abdominal operations certainly jus-

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tified the need for such an investigation to be reported.

Background for the study

The incidence of pulmonary complications following abdominal operations as reported in the literature has varied quite significantly among the various investigators, depending upon the thoroughness with which each series of cases was handled and particularly on the correlation of roentgen findings with clinical suspicion. It can be generally stated, with qualification and reservation, that the incidence of pulmonary complications following all operations is 2.5 to 3 per cent; following abdominal operations, 10 to 12 per cent; and following upper abdominal operations, 20 to 30 per cent.

These figures, as given by Kurzweg,[1] although a gross estimate, fail to differentiate between selected and unselected series of cases and fail to consider whether the investigators are partial toward clinical or roentgenographic aspects of complications. Statistics will usually be influenced when routine x-rays are obtained in the first few postoperative days.

Since this series of cases has been studied primarily for observation of pulmonary roentgenographic changes following cholecystectomy, regardless of clinical correlation, an analysis of previous authors' data will be limited to those series of cases of upper abdominal operations in which postoperative roentgen examination of the chest played a significant role in statistical evaluation of complications (see Table I). It is better to avoid some of the earlier series of cases for several reasons. Generally, the refinement of surgical and anesthetic technics, improved postoperative care, and the sometimes too liberal use of antibiotics have all contributed significantly to altering the incidence of postoperative pulmonary complications.

It is remarkable that almost no one has attempted to adequately describe the normal roentgen appearance of the postoperative chest since the series of 25 cases of upper abdominal operations reported by Muller, Overholt, and Pendergrass[2] in 1929, in which preoperative and 1-day postoperative supine films were made. With the exception of perhaps three studies,[3-5] in most of the literature reporting roentgenographic examinations performed postoperatively, the time interval and the frequency and consistency of examination were rather poorly controlled. In only one report[6] did the authors obtain consistent films after the sixth postoperative day.

Materials and methods

This series of cases consisted of 70 almost consecutive patients who had undergone either elective or emergency cholecystectomy. Five cases were eliminated because of inadvertent technical difficulties or inadequate roentgen examinations. Of the ten surgeons who had performed the operations, one sur-

TABLE I—REPORTS OF ROENTGENOGRAPHICALLY DETERMINED PULMONARY COMPLICATIONS FOLLOWING BILIARY AND GASTRIC SURGERY

Author and year	Number of cases	Type of operation	Day(s) of postoperative roentgenograms	Pulmonary complications	Remarks
Mimpriss and Etheridge, 1944[12]	100	Gastric	Within first few days and only if symptomatic	34%	Films on only 45 out of 100 No preoperative films
Stringer, 1947[8]	55	Gastric	Immediately After 4 hours After 24 hours	47%	First film supine, rest erect
Rudnikoff and Headland, 1951[4]	40	Biliary	Approximately fourth day Day of discharge	70%	Preoperative film within 24 hours of operation
Koch and Axén, 1951[6]	63 36	Biliary Gastric	Sixth to seventh day	38% 33%	Preoperative films done
Ekman and Odman, 1951[17]	123	Biliary	5 to 10 days	27%	Films taken in lateral recumbent position, and patient was fluoroscoped. Study primarily for pleural effusion
Thorén, 1954[9]	343 I-101 II-70 III-172	Biliary	Fourth or fifth day	 12% 27% 42%	Preoperative films done. Lateral films included. Study for value of physiotherapy. I-physiotherapy before and after; II-Only after; III-None
Brattström, 1954[3]	210	Gastric and biliary	All on fourth day Occasionally on second and sixth if symptomatic	50%	Preoperative films done; posteroanterior and lateral films, and fluoroscopy
Jones and Payne, 1955[10]	55 21 25	Gastric Biliary Other upper abdominal	First day Again within 48 hours	44% 19% 16%	Preoperative films done

geon operated in 35 of the total 65 cases reviewed. The length of operation varied from 30 minutes to 2 hours, with an average of approximately 1 hour. Almost all patients received Pentothal sodium induction with a combination of nitrous oxide and oxygen or cyclopropane, along with endotracheal intubation. Almost all patients were ambulated either the first or second postoperative day.

A preoperative chest film was taken on all patients, usually within 96 hours of operation. Postoperative roentgenograms were all taken with the patient erect, in an posteroanterior position, at a target distance of 6 feet. Films were taken on the third and seventh postoperative days on all patients, with an occasional follow-up film at a longer interval. An attempt was made to have the technic standardized so that almost all films were of comparable density.

The primary purpose of the roentgen examination was directed more toward the existence rather than the nature of pulmonary changes. Gross interpretations of linear, parenchymal, or vascular changes were noted, rather than subtle abnormalities that presented difficult diagnostic problems.

In addition to the roentgen studies, every patient was briefly questioned by the author on the days of the film examination as to previous respiratory history, unusual chest pain, dyspnea, or cough. The charts were examined for evidence of unusual elevations of temperature or leukocyte count. Each patient's physical examination, preoperative and postoperative auscultatory findings, clinical course, and suspicion of postoperative pulmonary morbidity were recorded from the progress notes of the intern and resident, who were unaware of the nature of the ensuing study.

Results and analysis

Of the 65 cases reviewed, there were 50 (77 per cent) females and 15 (23 per cent) males. The age distribution is shown in Table II.

TABLE II—FREQUENCY OF PULMONARY ROENTGEN CHANGES BY SEX AND AGE DISTRIBUTION

Age	No. females	No. roentgen changes		No. males	No. roentgen changes	
		3rd day	7th day		3rd day	7th day
10 - 19	1	1	0	0	0	0
20 - 29	7	3	0	1	0	0
30 - 39	10	3	2	2	1	1
40 - 49	10	6	3	6	1	1
50 - 59	6	5	4	3	1	1
60 - 69	14	8	7	2	0	0
70 - 79	2	2	2	1	1	0
Total	50	28	18	15	4	3

Preoperative chest films were considered to be within normal limits in 63 cases and abnormal in 2 cases, both of which showed pulmonary roentgen changes on both the third and seventh postoperative days. Of the 2 cases mentioned, both in 50-year-old women, one presented accentuated right hilar bronchovascular markings and subsequently demonstrated a right subsegmental atelectasis, while the other presented a right basilar linear atelectatic area and subsequently demonstrated bilateral multiple linear atelectatic areas (Fig. 1).

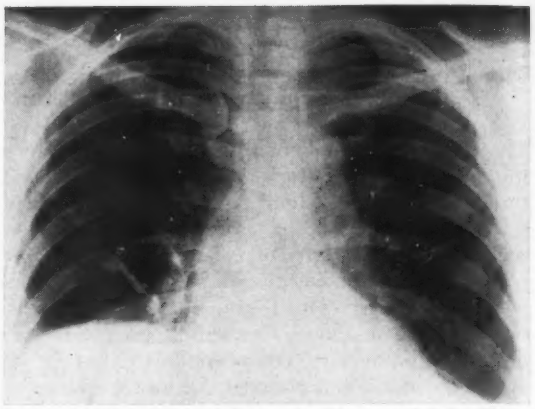


Fig. 1. Multiple bilateral linear densities associated with an elevated right diaphragm in a 51-year-old woman who demonstrated a right basilar linear density preoperatively. A cystic duct obstruction was present at surgery. The patient did not exhibit unusual respiratory distress on the third postoperative day.

There was a total of 32 (49.2 per cent) patients who showed pulmonary roentgen changes on the third postoperative day and 21 (32.3 per cent) patients who showed changes on the seventh postoperative day. All patients who showed roentgen changes on the seventh postoperative day also had changes on the third postoperative day. Therefore, 65.6 per cent of those with third-day changes had seventh-day changes. Of the females, 56 per cent (28 out of 50) showed pulmonary roentgen changes on the third postoperative day, while only 26.7 per cent (4 out of 15) of the males demonstrated changes.

Seven patients demonstrated more advanced pulmonary roentgen changes on the seventh day than they originally demonstrated on the third day. A few of these cases, followed at longer intervals, demonstrated residual changes at 3 to 4 weeks following surgery. The clinical correlation was not intensive enough to attempt to provide an explanation. Jahn and Olinger[7] reported on a series of 293 serial film examinations following phreniclasia and demonstrated that basal linear atelectasis may persist for 4 months to more than 5 years, and that with resumption of diaphragmatic motion, the opacities usually disappeared. Others have also apparently noted this.[8] Therefore, the linear densities which were thought to be atelectatic on the longer follow-up films did not appear to us to be of any clinical significance.

Regarding the nature of the pulmonary findings, 81 per cent (26 out of 32) of the third postoperative day changes were of a horizontal or oblique linear type. These linear densities, which ranged in thickness from 1 mm. to almost 1 cm., and which ranged in length from several millimeters to almost 10 cm.,

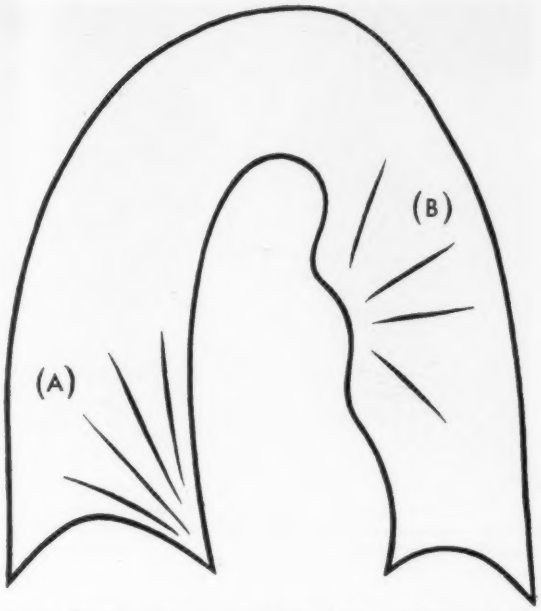


Fig. 2. Diagrammatic representation of the most frequent location of postoperative pulmonary linear atelectasis: (a) following the diaphragmatic dome to the mediastinum in a fanlike manner; and (b) radiating around the hilar regions in a sunburst manner.

were all considered to be linear atelectasis. Most of the oblique linear densities appeared to radiate from the cardiophrenic gutters toward the periphery in a fanlike fashion from the level of the diaphragms to the mediastinum (Fig. 2). However, many of the linear densities were found to be in rather bizarre directions. Nine per cent (3 out of 32) showed patchy or gross consolidative changes. Nine per cent (3 out of 32) showed significantly increased pulmonary vascularity, one of which was a frank pulmonary edema and was accompanied by linear atelectasis. Of the 32 cases which showed pulmonary roentgen changes on the third postoperative day, 15 involved the right lung field, 10 involved the left, and 7 involved both.

There was no operative or postoperative mortality in any of the cases for a period of 3 weeks after operation. Of the 4 patients having emergency operations, all of which were "surgically acute," 2 showed pulmonary roentgenographic changes on the third and seventh postoperative days, 2 showed changes only on the third day, and 1 did not show changes. There were 2 cases clinically suspected of having pulmonary infarction but only one, in my opinion, had roentgenographic findings compatible with this diagnosis.

If we were to consider only those cases in which the clinician was sufficiently alarmed by the patient's symptoms to order a roentgen study, only 8 (12 per cent) would have been studied by x-ray and, surprisingly, only 6 of the 8 cases would have had positive findings. In two patients with suggestive clinical evidence of pneumonia, roentgenographic studies had produced negative results.

An interesting by-product of the analysis showed that of the 24 patients (37 per cent) who demonstrated free peritoneal air beneath either or both diaphragms on the third postoperative day, 12 had accompanying pulmonary roentgen changes and 12 did not. None of the patients in this series demonstrated free air on the seventh postoperative day. Also, of the 23 patients who had operative and/or T-tube cholangiography, there was no demonstrable correlation with subsequent pulmonary roentgen changes.

Discussion

It has been estimated, with or without the support of roentgen findings, that atelectasis comprises nearly 90 per cent of all postoperative pulmonary complications. [3,9-11] Findings in this study are certainly in agreement with that figure if one considers that the 3 cases of patchy consolidative changes may have actually represented patchy atelectasis.[5,12,13] The nature of roentgenographic atelectatic change may range from the patchy variety mentioned by Swank and Smedal,[5] to the more common variety of platelike or linear atelectasis described by Fleischner and others,[14,15] to the most uncommon but easily recognized lobar type. It has been shown that roentgenographic manifestations of atelectasis vary, depending upon its stage of either resolution or collapse;[5,7,16] hence the frequent misdiagnosis of pneumonia. In this study there were at least 3 cases in which a poorly organized linear density resolved to a sharply defined linear density (Fig. 3).

Jones and Payne,[10] along with others,[17] were amazed at the lack of correlation between abnormal roentgenographic findings and the clinical symptoms. In the series reported here, only 12 per cent (8 out of 65) of the patients would have had x-ray studies if judged by clinical symptoms alone. Of the 8 patients, 2 failed to demonstrate any roentgenographic abnormality. Others report similar findings. Of the series of Koch and Axén,[6] only 16 per cent manifested symptoms, while there were 38 per cent who had pulmonary changes. Eckman and Odman[17] found respiratory morbidity in 17 per cent of their cases, while roentgen changes were demonstrated in 27 per cent.

Pneumonia as an early postoperative complication is uncommon.[18,19] There were only 2 cases in this series which could be considered as pneumonitic, based on both clinical and roentgen findings. Jones and Payne[10] stated that the most striking feature of their study was the complete absence of pneumonia and lung abscess. Wilson[20] went so far as to definitely state as a result of his investigation that "every postoperative pulmonary complication excepting that due to pulmonary embolism is, in the early stages of its career, an atelectasis." Many others are apparently in agreement, and feel that pneumonia is seldom primary in origin but develops in an area of atelectasis.[3,5,9,18,21-24]

Fig. 3. Successive postoperative films (left, third day, and right, seventh day) demonstrating a right basilar oblique patchy density, resolving to a more sharply defined linear density on the seventh postoperative day. The patient was moderately dyspneic on the third postoperative day but clinically negative on the seventh postoperative day. The roentgen changes were considered to be atelectatic.

It is not within the scope of this paper to review the various theories of the pathophysiology of atelectasis; so suffice it to say that there is considerable agreement[3,9,22,25-27] that the primary cause is probably a broncho-occlusive mechanism resulting from accumulated secretions induced by certain postoperative factors, a few of which are:

1. Altered postoperative pulmonary ventilation
2. Impairment of or inability to cough
3. Change in both the quality and quantity of the bronchial secretion
4. A reduction in the caliber of the bronchial tree.

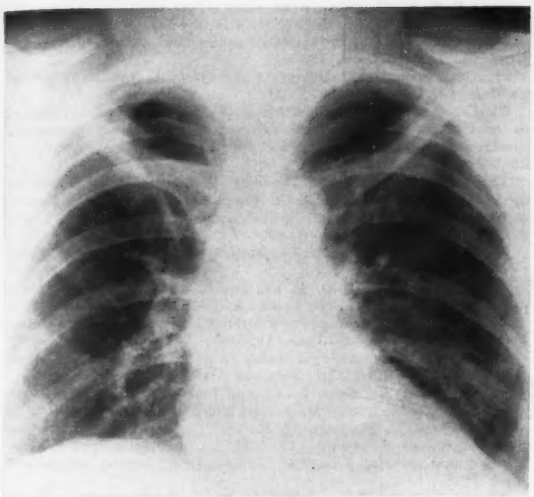
The reflex theory of de Takats, Fenn and Jenkinson[28] may explain the occurrence of some atelectasis, but the nature and pathogenesis of small atelectasis is not clear and warrants further investigation.[9]

The literature is overabundant with reports of the prevention and treatment of postoperative pulmonary complications.[9-11,29-33] Thorén's[9] interesting study demonstrated a considerable reduction in the number of postoperative pulmonary complications, both clinical and roentgenographic, when physiotherapy was used before and after operation. It might be well for some investigator to determine the value of osteopathic manipulative therapy in reducing the number of postoperative pulmonary atelectases as determined by roentgen study.

Despite considerable attention in the literature to pulmonary embolism, this is not a common early postoperative complication;[17,34] or at least it is not an early recognizable postoperative finding. In fact, some investigators have tended to disregard it completely.[9,17,35] There was only one probable case of pulmonary embolism in this series. The clinical manifestations of pulmonary embolism are so variable[36,37] and tend to overlap those of atelectasis to such a considerable degree[8,10,13,38,39] that the unqualified roentgenographic diagnosis of early postoperative embolism without clear-cut clinical correlation or frequent serial roentgenographic examination becomes extremely hazardous. It is possible that small linear atelectatic areas may sometimes be present as an indication of respiratory embarrassment, possibly associated with an early pulmonary embolus; but for the most part, early roentgenographic manifestations of infarct are considerably different in appearance.[40-43] It is well recognized that a great many cases of pulmonary infarction will not demonstrate roentgenographic changes.[37,40] It is remarkable how often the minimal symptom complex of dyspnea, cough, pleuritic pain, and elevation of temperature may accompany either atelectasis or infarction. This clinical similarity was especially noted when this series of cases was compared to another series of cases conducted in this same hospital by other investigators[44] who were primarily interested in the clinical diagnosis of pulmonary embolism.

The average diagnostic radiologist probably sees relatively few early postoperative chest roentgenograms, and a large percentage of those he does see will probably be the products of mobile apparatus used at the bedside. It is well to keep in mind that the diaphragms of early postoperative patients will almost invariably be elevated, the cardiac and mediastinal shadows more prominent, and the lower lobes somewhat increased in density. A good rule for comparison is that the very early normal postoperative chest film will resemble very closely that of an expiratory preoperative film taken on the same patient.[2]

It is almost safe to conclude that some degree of atelectasis without symptoms is a "normal" finding in postoperative films following major upper abdominal operations.[12] As was observed in this series



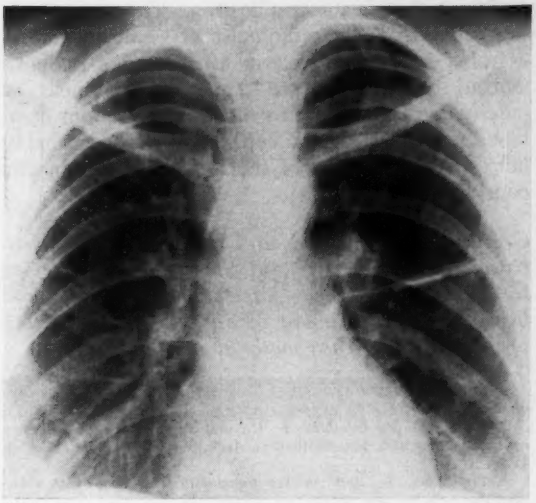


Fig. 4. Left, negligible pulmonary roentgen changes despite elevated diaphragm and moderate respiratory distress, on the third postoperative day in a 52-year-old hypersthenic woman. Right, bilateral linear atelectatic areas of the midpulmonary fields in a 26-year-old asthenic woman who was completely asymptomatic on the third postoperative day. The diaphragm was not significantly elevated. The roentgenographic changes were completely gone on the seventh postoperative day.

of cases, the diaphragms were almost always significantly elevated during the third postoperative day, regardless of associated abnormal roentgen findings (Fig. 4). There appeared to be no correlation between what seemed to be significant diaphragmatic elevation with the frequency and intensity of the abnormal pulmonary roentgenographic findings on either the third or seventh postoperative day. Therefore, it is apparent that diaphragmatic elevation would, in most instances, not be of any definitive value in differentiating small atelectases from areas of pneumonia or infarction. Finally, pulmonary complications may be frequently but not always confirmed by x-ray examination.[19]

Although several early investigators attributed the frequency of postoperative pulmonary complications to the types of anesthesia employed, it is now generally considered to probably be of little or no significance, provided the usual forms of anesthesia are delivered by competent anesthesiologists.[13,35,45] Unusual length of surgical procedures may contribute to pulmonary complications.[29] Obese persons and those afflicted with chronic bronchial conditions, including heavy smokers, are probably more disposed to the development of pulmonary complications.[9,29] Males are generally more affected than females, in a ratio of 2 or 3 to 1.[19,35] This series of cases does not bear this out, but the number of males is probably too small to be of statistical significance.

Aspiration pneumonitis is considered a rare postoperative complication today.[38] Although this series of cases failed to produce one which was clinically or roentgenographically suspected of having aspiration pneumonitis, this diagnosis must be kept in mind. Postoperative patchy atelectasis with suspected superimposed pneumonitis should alert one to the diagnostic possibility of chemical pneumonitis due to aspirated gastric contents, either silent or otherwise.[46-48]

Summary

About half of an unselected series of patients undergoing cholecystectomy demonstrated pulmonary roentgenographic changes on the third postoperative day, and about two thirds of the latter cases demonstrated changes on the seventh postoperative day. The great majority of pulmonary roentgen changes consisted of horizontal or oblique linear densities and occasionally patchy consolidative areas, all of which probably represented atelectasis at various stages of collapse or resolution.

These linear and patchy changes may present two major roentgen differential diagnostic possibilities, pneumonia and pulmonary infarction, both of which are uncommon early postoperative pulmonary complications following upper abdominal operations. In regard to these linear or patchy densities, the diagnosis of atelectasis, pneumonia, or infarction cannot usually be based on a single early postoperative roentgenogram, since it is obvious that in many cases the three disorders may be indistinguishable roentgenographically. It has been shown that there is a large area of symptom overlapping in the clinical manifestations of atelectasis and pulmonary infarction.

Only 8 patients (12 per cent) in this series of 65 cases would have been studied by x-ray because of the concern of the attending physician; of these, 2 patients failed to demonstrate any gross abnormal pulmonary roentgenographic change. The majority of patients who demonstrate pulmonary roentgen findings of a linear or patchy nature in the first postoperative week will not be significantly symptomatic to be clinically considered as a postop-

erative pulmonary complication. Therefore it is suggested that the radiologist or clinician should exercise extreme caution in attributing a patient's morbid clinical state to demonstrated linear or patchy densities. Serial roentgen examinations combined with close clinical correlation would probably help to resolve some of the differential diagnostic considerations.

Gratitude is expressed to Drs. Robert R. Rosenbaum, Philip Shtasel, and Samuel Santaspirt for their expert guidance and teaching, and to members of the Department of Surgery for permitting me to use their case material for this study.

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Pruritus ani: Etiology and therapy

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Pruritus ani is a perianal irritation capable of producing a violent nettling, intolerable burning, and itching syndrome which prevents rest and relaxation, inciting nervousness and irritability so disrupting to the nervous system that in some cases it has led to emotional instability. The severity of this ailment, the difficulty of obtaining a comprehensive diagnosis, and, quite often, the failure of obtaining satisfactory and lasting results place pruritus ani in a number one position on the list of proctologists' problems.

Pruritus ani simply means intense itching at the anus. It is not a disease, only a symptom. Its etiology may be found to be local, singular, and simple, or it may be remote, multiple, and quite complex. The therapy, accordingly, may require only simple measures to bring about early and lasting relief, or, as is true in many cases, it may be complex because of the many factors which may be found as background for this localized symptom.

Perianal itching may be incited by one or more of some fourteen causative factors. These are herein divided into two groups, seven of which are classified as local or primary and seven termed remote or contributory. The remote disorders which should be considered are so many and varied that a proctologist cannot limit his search to his own specialty field. He must enter the realm of the internist, seeking the assistance of laboratory tests which help to evaluate liver function, fecal and gastric analysis, blood chemistry, and so forth; in fact, he must use any or all tests which may bring to light the background for an unbalanced biochemical state.

Since the search for contributing factors may encompass the entire body, and the disorder may be keyed with dysfunction of very remote organs, pruritus ani cannot be classified entirely as a proctologic complaint. However, the tormented patient comes to the rectal specialist because he feels certain that it is within the proctologist's ability to give him assistance. It therefore becomes the rectal specialist's problem to see that a proper diagnosis is effected, however difficult.

Great care and caution should be exercised in dealing with pruritus ani. Too many proctologists are willing to assume that the tissue disorders discovered on the first examination are the sole cause for the patient's complaint and offer assurance that surgical measures will be conclusive. Sadly enough, in many cases both patient and doctor are disheartened with the results. When this happens the doctor either loses his patient or he must start a search of the other thirteen possible causes. If the patient remains with the doctor, his confidence is doubtless somewhat shaken and he is less likely to cooperate if further examination becomes quite complicated. It is therefore a good policy to acquaint all pruritus patients with the seriousness of their condition and explain the possibilities of other local or systemic backgrounds before an operation is performed, even though a cryptitis or other tissue disorder is present, or if a plausible factor does seem apparent.

It is not my purpose to recommend an exhaustive study of all possible causes if a local cause should be confirmed early in the examination. Treatment should be directed to any apparent contributor as soon as such is apprehended. However, since other factors may be coexistent, the physician must proceed with alertness. He must acquaint the patient with all the possibilities, preparing him for continuance of the search should results not be entirely satisfactory and lasting. When there is a complete understanding between the doctor and his patient, the patient does not resent additional

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laboratory studies should such become necessary.

In the treatment of pruritus ani, I feel that the main faults lie in the doctor's failure to realize how complicated the condition usually is, and in his inadequate explanation to the patient. Success is dependent upon good teamwork, and the doctor-patient cooperative team is certainly essential in the handling of pruritus ani.

Since the advent of our specialty, pruritis ani has been frowned upon and shunned by proctologists. There has never been a panacea and, of course, never could be. It is a wearisome task to run down all the contributing factors. The ailment is neither crippling nor fatal, yet it may give rise to embarrassment caused by the sudden, stablike, painful itch which commands the patient into action for relief. This series of needling and sudden itching seizures so shocks the nervous system that the patient's disposition and general health are definitely affected. It is difficult to say whether the sick system is producing the symptom or the symptom is producing the sick system.

For years I have studied the pruritus ani problem. The longer I search, the more complex I find it to be; yet the grateful patients have been a stimulant to bring before proctologists this subject as simply yet completely as possible. For this purpose I have prepared the outline which appears in the Summary, to enable the physician to ascertain at a glance the fourteen etiologies, any one of which is capable of inciting the itch-scratch cycle. In the following discussion each cause is segregated, together with some suggestions for therapy. The reader will note that special emphasis is placed upon three etiologies. This is done because their percentages are paramount and should be among the first to be considered in all cases. These three are internal parasites, intestinal stasis with altered pH, and liver dysfunction resulting in an overabundance of histadine which is converted into histamine.

How frequently the patient comes to us complaining of "itching piles." In the layman's mind piles and itching are synonymous. Of course hemorrhoids do not itch and many patients are afflicted with pruritus ani without having hemorrhoids. When both are present it must be realized that we are dealing with two entities. It is true that the folds of external redundancies form an excellent harboring site for monilia and parasitic ova. Surgical treatment is necessary but cannot be conclusive.

Seven local or primary etiologies

1. **Pathologic tissues** • These can include skin tabs, condylomata, fissures, hemorrhoids, fistulas, and infected crypts. Treatment consists of adequate surgical correction.

2. **Internal parasites** • One infecting agent is oxyuris vermicularis (Enterobius vermicularis, pinworm, or seatworm).

In one method of treatment, santonin and calomel, 1 to 3 grains, are given on three successive nights. A nongreasy diet should be followed during this course of medication.

In another treatment form, piperazine salts, citrate, phosphate, or adipate, are the drugs used; they are available as a palatable syrup, tablets, or wafers to be chewed. The drug is most effective when taken before breakfast and followed by a glass of water; this carries it to the cecum and colon, the habitat of the worms. Therapy is continued for 7 consecutive days. Employing the following dosage schedule, Brown[1] cured 58 of 60 patients.

DAILY DOSE OF PIPERAZINE USED IN THERAPY OF ENTEROBIASIS[1]

Patient's weight	Piperazine citrate syrup	Piperazine hexahydrate
Up to 15 lb.	2.5 cc.	0.25 gram
16 to 30 lb.	5.0 cc.	0.50 gram
31 to 60 lb.	10.0 cc.	1.0 gram
Over 60 lb.	20.0 cc.	2.0 grams

An effective, inexpensive therapeutic agent for the treatment of enterobiasis is gentian violet in enteric-coated tablets. Administration for 10 consecutive days is effective and relatively well tolerated; recently a 7-day treatment program was reported as successful.[1] The usual adult dose of gentian violet is 0.065 gram (1 grain) three times a day before meals. Children are given 10 mg. per year of age; thus a 10-year-old would receive a total of 100 mg. a day, or 33 mg. (½ grain) three times a day.

Oxytetracycline is effective but expensive, especially when the treatment of a whole family is necessary. The usual adult dose is 2.0 grams a day for 5 to 7 days.[1]

Perhaps the newest of remedies is pyrvinium pamoate. This is given in a single dose, 1 teaspoon per 22 lb. body weight and should be repeated in 7 days.

The treatment of a person harboring pinworn is frequently unsatisfactory if other infected members of the household are untreated and serve as a source of reinfection. It is recommended, therefore, that all members of the household be examined by the Scotch tape swab technic and that those infected be treated simultaneously.[2]

Ascaris lumbricoides (the ellworm or roundworm, a common worm resembling the earthworm) is found in the small intestines, especially in children. It causes colickly pains and diarrhea.

Piperazine salts are also effective in ascariasis. A single dose cures 75 to 85 per cent of the infections, and treatment on 2 consecutive days will eliminate approximately 95 per cent of the infections.[1]

Since the presence of food in the digestive tract has little, if any, effect on the activity of piperazine

against ascarides, the piperazine may be given at any time. No special dietary precautions are necessary, and purgation is not required. The ascarides usually are passed on the first, second, or third day after treatment, and they are immobile and relaxed though not dead. In vitro studies indicate that solutions of piperazine produce a state of relaxation and narcosis of the worms after a few hours.

DOSAGE SCHEDULE FOR ONE- AND TWO-DAY TREATMENT OF ASCARIASIS WITH PIPERAZINE[1]

Patient's weight	Daily dose as citrate syrup	Daily dose as hexahydrate
30-50 lb.	20 cc.	2.0 grams
51-100 lb.	30 cc.	3.0 grams
Over 101 lb.	35 cc.	3.5 grams

One effective ascaricide which has been used extensively is hexylresorcinol. With this agent it is essential to make certain that the intestinal tract is empty when the drug is given, or it will combine with food and have little action on the parasite. Therefore it is advisable that the patient eat only a light supper the evening before treatment. The following morning, breakfast should be omitted and the medication given early. The dose of hexylresorcinol is 0.1 gram for each year of age up to 10, the adult dose being 1.0 gram. Hexylresorcinol is available in pill form and should be swallowed with a little water; it is essential to see that the pills are swallowed whole for if they are chewed they cause an annoying superficial burn. The patient must refrain from eating for 5 hours after treatment. A magnesium sulfate purge is given on the following morning to hasten the expulsion of the injured and dead worms.

Not all ascarides are passed immediately after treatment; they may continue to appear in the stool for as long as 10 days. Therefore, the post-treatment stool examination should be delayed until 2 weeks after treatment. If eggs are still present, a second treatment is required. Approximately 90 per cent of all the ascarides are removed with a single treatment. After two such treatments, from 93 to 98 per cent of the patients can usually be considered cured.[1]

Taenia saginata is the beef tapeworm. Diphyllobothrium latum is a tapeworm for which crustaceans and fish are hosts. Hymenolepis nana is a dwarf tapeworm found in the adult form in the human intestines, frequently causing a local disturbance. For treatment of these, quinacrine hydrochloride is considered the best drug because of its great efficacy and relatively low toxicity. A second choice is oleoresin of aspidium (male fern).

The preparation of the patient is important in treatment for tapeworm. On the day preceding treatment, the patient should be placed on a liquid diet, and supper should be omitted except for black coffee, tea, and water. A soapsuds enema should be given in the evening. The following morning the patient should remain in bed, omit breakfast, and take the anthelmintic. Patients, either adults or children, who vomit quinacrine or aspidium may be successfully treated with medication given by duodenal tube. Two hours after the last dose of drug a saline purge should be administered and followed by a glassful of water. If the worm is not removed by purgation or if the head is not recovered, a soapsuds enema should be given as the worm or scolex may be lodged in the large intestine. Toilet paper used by the patient should not be placed in the bedpan as it makes the search for the tapeworm head very difficult.

If large portions of the worm are recovered and the head is not found, the stool should be free of eggs and segments for 6 months before one can be sure that the treatment was successful. The head may have been digested, lodged in the large intestine, or not recognized in the stool examination following therapy; therefore, failure to find the head does not necessarily mean failure of cure.

The total adult dose of quinacrine is 0.8 gram. In order to prevent vomiting it may be given as 2 doses of 0.4 gram with an interval of ½ hour between, or 0.2 gram every 10 minutes. The children's dose is 0.4 gram for those of 40 to 75 pounds and 0.6 gram for children of 76 to 100 pounds.

In the dosage employed in tapeworm therapy quinacrine may produce nausea and vomiting. Transient dizziness and yellowish discoloration of the skin occasionally develop.

Magath and Brown[3] report success with an emulsion consisting of 6.0 grams or 6.0 cc. of oleoresin of aspidium (Brown[1] uses 5.0 cc.), 8.0 grams of acacia, and water to make up to 60 cc. One dose of 30 cc. of the emulsion is given early in the morning, followed by an additional 30 cc. 1 hour later. The total dose for children is 4.0 cc. of the emulsion per 10 pounds of weight. Therapeutic doses of oleoresin of aspidium may cause, in varying severity, headache, vertigo, gastroenteritis, abdominal pain, diarrhea, nausea, vomiting, and temporary changes in vision.

Although quinacrine hydrochloride and oleoresin of aspidium are effective therapy for tapeworms in children, because of the frequency of vomiting among children given these substances, the cure rate is low. Iodoalphionic acid, which is used for the visualization of the gallbladder, has been reported by Shookhoff and Sterman[4] to be effective in these three tapeworm infections. They gave 3.0 grams of this agent to children weighing 40 pounds or less and suggest 6.0 grams for childern of 100 pounds. They consider it the drug of choice for infected children as it is well tolerated by them and, unlike quinacrine and oleoresin of aspidium, does not cause vomiting.

Another infecting agent is Entamoeba histolytica. The choice of therapy for intestinal amebiasis depends upon the severity of symptoms. Mild and asymptomatic infections may be treated with one of the halogenated oxyquinoline compounds such

as chiniofon, diiodohydroxyquin, or iodochlorhydroxyquin, or one of the arsenicals, carbarsone or glycobiarsol. Diiodohydroxyquin, because of its relative freedom from side effects, is widely used; it is given orally for 20 days at the rate of 0.65 gram three times a day. Children weighing 40 to 75 pounds may be given 0.65 gram twice daily, and children under 40 pounds may receive 0.32 gram three times a day.

For acute amebic dysentery, emetine and oxytetracyline should be employed. The emetine is given subcutaneously in doses of 1 mg. per kilogram of body weight per day. This usually results in cessation of the dysentery in 3 to 5 days, at which time the medication should be discontinued. Emetine is remarkably effective in controlling dysentery but is not effective in eradicating the infection. Oxytetracycline is continued for 10 days, giving 40 mg. per kilogram of body weight per day. On conclusion of this therapy, a course of diiodohydroxyquin, followed by a course of carbarsone, should be given. Carbarsone is given for 10 days, 0.25 gram three times a day. Stools should be examined several times during the 6 months after treatment.

For amebic hepatitis chloroquine phosphate and emetine are effective, and as the former is less toxic it is the drug of choice. Erythromycin is also of value in hepatic amebiasis. For acute hepatitis or liver abscess 0.25 gram of chloroquine two or three times a day for 3 weeks is recommended. Children of 40 to 75 pounds receive 0.25 gram twice daily for 2 weeks. With the exception of occasional pruritus and gastrointestinal discomfort, chloroquine therapy is usually without side effects. If emetine is used, the total dose is 10 mg. per kilogram of body weight over a period of 10 days. The pulse and blood pressure should be taken before each injection. All cases of amebic infection of the liver should also receive a course of treatment for intestinal amebiasis, as chloroquine or emetine cannot be depended upon to clear up the intestinal infection.

For rectal Trichomonas infections, treatment includes carbarsone suppositories, iodochlorhydroxyquin inserts, and antiseptic powders. Acidifying the rectal ampulla is a necessary adjunct.

3. **External parasites** • For Trichomonas vaginalis, treatment includes iodochlorhydroxyquin inserts and insufflation, diidohydroxyquin, and ricinoleic acid. For acid douching, 2 tablespoons of vinegar to 1 quart of warm water are used.

For treatment of scabies, a 25 per cent emulsion of benzyl benzoate is used. For pediculus pubis or corporis, isobornyl thiocyanoacetate lotion or gamma benzene hexachloride is useful.

4. **Mycotic infections** • The yeastlike fungi causing infections in the anal area are Candida albicans (Monilia), Candida immitis, Trichophyton (dhobie itch), Epidermophyton, and Cryptococcus neoformans (Torulopsis histolytica).

For topical therapy, Castellani's modification has made the popular Whitfield's ointment less irritating. One formula is:[5]

Rx	Phenolis	gr.v
	Acidi salicylici	gr.xv-xx
	Acidi benzoici	gr.xv-xxx
	Petrolati	qs. to oz.1

Another formula which gives good results is[5]:

Rx	Prec. sulphur	gr.xv-xxx
	Acidi salicylici	gr.xv-xxx
	Petrolati	qs. to oz.1

Other agents for topical use are carbolfuchsin (alcohol) solution, propionate-caprylate (fatty acid) compound, and undecylenic acid (ointment and powder).

For oral therapy, griseofulvin tablets, 250 mg., are given in doses of one to four tablets daily, depending on weight. Nystatin, 500,000 I.U. in oil, is given in doses of one to three tablets daily; this may be doubled if necessary.

5. **Allergies** • These include food idiosyncrasies, including "gormandizing" and eating of highly seasoned foods; drugs, such as Novocaine, quinine, belladonna, resorcinol, and arsenic; an overabundance of histamine; and dermatitis venenata (contact dermatitis).

Allergic foods can be eliminated by trial and error; allergies are so variable that each case is a law unto itself. Foods most usually found to be offending are tomatoes, shellfish, strawberries, chocolate, nuts, highly seasoned foods, and condiments. A high carbohydrate diet quite frequently is at fault. This is especially true in cases of fungous infections. In cases of contact dermatitis, withdrawal of the offending allergen is necessary.

6. **Altered biochemistry** • A high pH stool can be caused by pathologic intestinal flora, colonic stasis, faulty diet, alcoholism, histamine, intestinal toxemia, or Streptococcus faecalis infection.

When aciduric bacteria predominate in the colon, the growth of putrefactive organisms, such as Escherichia coli, is lessened. The growth of yeastlike fungi, such as Candida albicans, is likewise inhibited. There is evidence that these organisms produce substances which are irritating to the anal canal and perianal tissues and which seem to be responsible for the itching sensation in some cases of pruritus ani. If this hypothesis is correct, any change of the intestinal flora which would acidify the rectal discharges of these patients should result in relief of symptoms.[6]

Granet[7] has pointed out that the secretions of the mucosa of the rectum are markedly alkaline in many patients with pruritus ani. He has suggested that the lowering of the pH of the rectal discharges may be the important factor responsible for periods

of remission. By utilizing nitrazine paper, he has repeatedly found that the fecal material and secretions of the rectum of these patients are strongly alkaline. In these cases, the pH is about 9 and, after successful treatment, the pH is reduced to 6 or 7.

Slocumb[8] evaluated disturbances of the acid-base balance in a series of 105 cases of pruritus ani. He summarized by stating that the findings were of a chemical irritant, alkaline in reaction, metabolic in origin, and favorable to a correlation of the mucosal pH and bacterial flora.

Cannon and McNease[9] concluded that the pH of the colon was a decisive factor in the promotion of the aciduric flora. Sokoloff[10] stated that as a result of the low absorptive activity of the mucous membrane, the bacterial flora becomes the decisive factor, and the functioning of the colon and its protective acidity depends, to a large part, on the presence of acidophilic flora. In ulcerative processes of the lower bowel, this investigator observed a constant disappearance of the aciduric flora and a shift of the contents toward alkalinity.

Many diversified therapies are employed in changing the pH of the intestinal flora. A suspension of Lactobacillus acidophilus in a petroleum base is worthy of use. Brooks[6] used malt extract of barley which had been neutralized with potassium carbonate and concentrated to a heavy syrup in infant and adult feeding to combat constipation. Its action appeared to be dependent upon its property of aiding production of aciduric bacteria in the distal portion of the colon and consequent acidification of its contents.

Slocumb[8] observed that when the excessive alkalinity of the intestinal flora was corrected by an acid-ash diet and local therapy, the bacterial flora returned to normal and the pruritus subsided.

In one text[5] there is a report of acid-ash diet therapy in a number of private patients; this diet was found to be a valuable adjunct in the treatment of pruritus ani. Many of these patients were referred to a gastroenterologist for gastric analysis, and reports disclosed an achlorhydria.

This same text gives an acid-ash diet used in the treatment of 125 cases of pruritus ani. The diet and the results are as follows:

ACID-ASH DIET FOR PRURITUS ANI

Allow: *Soups:* Milk, rice, noodle or barley soup, meat soups
Meat or fish: All kinds, two servings daily
Vegetables: Corn, mushrooms, asparagus, peas, onions, radishes, turnips; potatoes may be used sparingly
Desserts: Cake, prunes, plums, cranberries, custard, rice, tapioca and cornstarch puddings, ice cream. Apples and pears may be used sparingly
Breads: All kinds, crackers, cookies
Cereals: All kinds
Beverages: Prune and plum juice, tea, coffee, cocoa, milk
Miscellaneous: Butter, cream, cheese, jam, mayonnaise, eggs, rice, noodles, barley
Restrict: All fruit and vegetables not listed above.

Sample menu:
Breakfast
Prunes
Oatmeal—cream
2 eggs
Whole wheat toast, butter
Coffee
Dinner
Beef bouillon
Roast lamb, large serving
Buttered rice
Asparagus tips
Whole wheat bread, butter
Bread pudding
Tea
Supper
Chicken broth
Beef patty
Stewed corn
Whole wheat bread, butter
Plums
Milk

In addition, a capsule of glutasin (glutamic hydrochloride, gr. v; pepsin, gr. ¼) is given before each meal and at bedtime, and a gelatin suppository containing betalactose, 20 grains, is inserted into the rectum upon retiring.

RESULTS IN TREATMENT OF 125 CASES OF PRURITUS ANI

	Cases
Symptom-free—3 months or more	84
Showing improvement—3 months or more	37
Not improved	4
Showing recurrence during treatment (usual reason—not following instructions)	21
Showing allergic manifestations and treated	11
In which fungi were demonstrated	16
Showing parasites—usually pinworms	4

Histamine is formed by the putrefactive bacteria in the lower intestinal tract. Thus, the histamine, indole, and skatole overabundance, which is consistently found in the feces in intestinal stasis, tends to lead back to the altered pH etiology. The normal flora should consist of approximately 70 per cent of the protective organisms, such as Bacillus acidophilus and Bacillus bifitis, and only 30 per cent of the putrefactive groups. In colonic stasis, these percentages are quite frequently reversed, allowing for a high pH and excessive putrefaction.

Murray[11] states that all cases of pruritus ani which he observed were caused by streptococcic infection, of which the Streptococcus faecalis was the predominating organism. This was more recently confirmed by Bassler and Connors.[12] Allingham,[13] however, insisted that pruritus was due to some pathologic or functional origin. Lockhart-Mummery[14] stated that a fibrosis of the peripheral nerve endings of the anal and perianal region was the etiologic factor. Pennington[15] was of the opinion that pruritus was due to deposits of an inflammatory exudate in the tissues having a lowered degree of vitality. There is some basis of truth in all of these varied opinions; yet, since these opinions differ so greatly, no one of them is likely to be entirely right.

7. **Itch-scratch syndrome** • This may involve uncleanliness, perianal skin hypertrophy, and neurodermatitis.

Uncleanliness is a common factor, especially in cases with intestinal stasis where there is likely to be an abundance of indole, skatole, and histamine, and a predominance of Escherichia coli, Bacterium aerogenes, and other putrefactive bacteria. These exudates and infectious materials, when accumulated on the excoriated surface of the perianal zone, most certainly contribute to inflammation.

The itch-scratch cycle promotes skin hypertrophy and may confirm the opinion of Lockhart-Mummery[14] concerning fibrosis of the peripheral nerve endings. Because histamine is generated principally in traumatized tissues, the itch-scratch cycle perpetuates its existence in this region.

Neurodermatitis is secondary to nerve shock and can be local as well as general. The perianal region is perhaps the most sensitive reflex center and, therefore, it is easy to conceive the possibility that such reflexes caused by intense itching can be an etiology.

Topical therapy would include ointments containing hydrocortisone or amino-acid ointment, which would break the itch-scratch cycle.

Seven remote or contributing etiologies

1. **Remote infections** • These would include bronchiectasis, pyorrhea alveolaris, and infections with such bacilli as staphylococci, Escherichia coli (Bacillus coli), Streptococcus faecalis, and gram-positive diplococci.

Bronchiectasis, a condition wherein copious amounts of disintegrated cells and bacteria are constantly swallowed, most certainly can be given as a contributing agent. These disintegrated tissue cells add to the production of histidine from which histamine originates. Pyorrhea alveolaris is often a source of blood-borne infection which stagnates in inflamed tissue and incites toxic inflammation (one that is caused by poison, such as a bacterial product). Gram-positive diplococci, staphylococci and Streptococcus faecalis are commonly found in the normal flora. However, they can become pathogenic in patients with lowered resistance, especially when there is a high pH environment. Escherichia coli and Streptococcus faecalis can be true pathogenic factors when their abundance is capable of overthrowing the normal flora pH. Their toxins are more devastating in devitalized patients, producing an exudate which chronically irritates the perianal skin.

A competent laboratory capable of studying the sensitivity of bacteria to the various antibiotics should be consulted before treatment is instituted.

2. **Blood dyscrasia** • This could be anemia, lithemia, cholesterol imbalance, or an increase in bile salts.

Both pernicious and secondary anemia contribute to anoxia resulting from decreased hemoglobin and number of erythrocytes. Stagnant anoxia causes lowered tissue resistance and decreases the vitality of the skin. Lithemia (increase of urates in the blood stream) must also be considered as a correlate often existing in pruritus ani. A cholesterol imbalance, either elevated or lowered, has been given by several authors as a finding in most pruritus cases; it must therefore be considered in the appraisal of systemic backgrounds. Increased bile salts in the blood stream in cases of hepatic obstruction produce a generalized pruritus and must not be omitted from the over-all picture.

The usual treatment for these various conditions should be employed in every case, and it may be found to alleviate the pruritus ani.

3. **Hormone and endocrine imbalance** • This may be thyroid dysfunction, hypoadrenia, estrogenic and androgenic imbalance, senile and menopausal influence, and urticaria subcutanea.

Hyperthyroidism causes hypersensitiveness. Hypothyroidism contributes to sluggish digestion, intestinal stasis, and retarded metabolism. This slowing down contributes to excessive intestinal putrefaction (indole, skatole, and histamine) from which an irritating exudate originates.

The adrenal medullary hormone, epinephrine, may have both pressor and depressor effects upon the circulation. The cortical hormone affects digestion, circulation, tissue metabolism, renal function, and resistance to stress. Loss of adrenal cortex function results in profound changes in biochemical and physiologic functions. In adrenal disease there is increased blood urea, muscular weakness, and decreased resistance to shock (cold, mechanical, and chemical). Deficiencies may be related to pruritis ani. Hypothyroid and hypopituitary function are correlated with apocrine gland function, a source of cholesterol excretion in the perianal zone.

Influence of estrogenic and androgenic imbalance contributes to irritability or apathy, depleting sphincter and tissue tonus. Senile and menopausal influence, in respect to tissue tone and reflex intolerance, must be considered.

The swelling caused by tissue edema in the perianal zone often produces urticaria subcutanea; this is kept in motion by constant trauma from scratching, resulting in a continuous state of edema.

Adequate hormone therapy and, in cases of urticaria subcutanea, topical application of hydrocortisone in an acid-mantle vehicle often will arrest this cycle. Amino-acid ointment has proved efficient. Sedatives and tranquilizing agents also play a role in arresting the itch-scratch cycle.

4. **Systemic diseases** • These include achlorhydria, atony, Hodgkin's disease, diabetes, and organic reflexes.

As reported by Harwick and Bacon,[16] achlorhydria was found to be a constant symptom in all pruritus ani cases in which a gastroanalysis was made. Atony, from whatever cause, contributes to the severity in that there is a loss of tone and

vitality, especially in the diseased area. According to Gradwohl,[17] pruritus is a symptom in at least 10 per cent of cases of Hodgkin's disease. Diabetes is conducive to pruritus ani, especially when there is a local colonization of Monilia, since fungi live and maintain a vigorous growth when feeding on sucrose. The sugar contained in the sweat of the diabetic patient promotes the luxurious growth. Also, the inability of such patients to heal properly allows the abraded surfaces to remain open for further fungous invasion. Prostatitis, cystitis, uterine and ovarian tumors and cysts, urethral stricture, and phimosis can all produce reflexes to the perianal zone and must be appraised when there is the least doubt as to the true cause.

Should any of these conditions coexist with pruritus ani, it is important that they be treated in the usual manner concurrently with the treatment of pruritus.

5. **Vitamin deficiencies** • Avitaminosis impoverishes tissue tonus and vitality. Sphincter tonus is usually lacking in pruritus ani. Vitamin B complex deficiency produces vascular dystonia (lack of tone in wall of artery and vein), which allows for increased congestion and blood stagnation. Vitamin A and D deficiencies lower resistance to bacterial invasion. Vitamin A is necessary for healthy epithelium and vital for normal skin continuity. Vitamin C is dissipated by shock and is the first to fall below the normal level in all diseases and infirmities. It is most essential for healing. Repair to the constant traumatic damage in this zone falters if vitamins A and C are below normal. Vitamin therapy should never be overlooked.

6. **Psychosomatic factors** • These include worry, fright, overwork, sedentary occupation, heterosuggestion, autosuggestion, psychosis, melancholia, and associated maladies. It is a fact that worry, fright and sedentary occupation affect biochemical balance. A patient, seeing someone else scratch his face or head, is reminded of his own problem and is alerted to satisfying his nerve stimulus by scratching. Fear of having to scratch in the presence of others incites his need for this satisfaction. He is therefore in constant dread and ever mindful of his situation. Sleeplessness and irritability are forerunners of psychosis and melancholia. Repressed sexual urge and sometimes masturbation are considered as associated with pruritus ani, perhaps even as causative factors.

7. **Altered liver metabolism** • This may be manifest as cholestasis, cirrhosis, histidine or histamine disorders.

I wish to elaborate on this topic since many of us do not consider the consequence of cholestasis as influencing pruritus ani. Incipient liver disease is insidious and may not be recognized; hence there is a possibility that early hepatic disturbances may promote an advanced symptom such as pruritus resulting from an exudate rich in histamine.

Cholestasis is caused by either intrahepatic or extrahepatic obstruction. Bile stasis results from both, causing enlargement of the liver and inflammation of the portal tracts, and also the dilatation of the bile canaliculi which contain bile plugs. Eventually, bridges of cholestatic tissue connect the central canals. These changes are more prominent in the center of the lobule. In the presence of severe periportal inflammation, bile stasis is conspicuous on the lobular periphery by kinking the ductules around the limiting plate. The perilobular and intralobular ductules contain larger bile plugs or microcalculi and are often dilated. Rupture of the biliary canaliculi adjacent to cells with feathery degeneration or of the ductules results in bile pigment free in the tissue (bile lakes), around which a few scavenger cells and segmental leukocytes accumulate. Thus, hepatic cell necrosis, whether intrahepatic or extrahepatic, exists, and the systemic reaction is practically the same. The severity is dependent upon the degree of obstruction or the amount of liver tissue obstructed. In cholestasis, there is inflammation and necrosis of the hepatic cells. There is a rise of bilirubin and bile acids in the serum, this being partially the result of regurgitation. Part of the bilirubin elevation is the result of bile acids in the regulation of the salt and water balance of the body, and the biliary excretion of alkaline substances influences the acid-base balance. Large amounts of free bile acids are found in hepatic diseases.

Histidine is formed from the protein of broken down or necrosed cells and tissues. Thus, in liver obstruction, there is an overabundance of histidine.

Histamine is formed in the gut by bacterial decarboxylation of histidine. It is also formed in injured tissues and apparently is continually formed in small amounts in normal tissues. Allergic reaction appears to be related to the explosive liberation of histamine caused by entrance of the sensitizing substance into the tissues. Its excessive liberation may be related to traumatic shock. Histamine markedly depresses blood pressure, and large doses may cause extreme vascular collapse. Histamine strongly stimulates the secretion of both pepsin and acid by the stomach. An enzyme, histaminase, which destroys histamine, is widely distributed in the body, with the exception of the lungs. This may account for the accumulation of histamine in this organ. Histamine increases portal pressure while it lowers systemic blood pressure. It increases bile production and stimulates smooth muscle in general and contracts the gallbladder while occasionally relaxing the sphincter of Oddi, the end result being a cholekinetic effect.[18]

The action of histamine, causing systemic hypotension, can also encourage atonia, which is a common finding in pruritus cases. Since itching is the prime symptom of which these sufferers complain, and histamine is the prime agent for producing this nervous reflex, it is, in my mind, of great importance to study the origin of an overabundance of histamine, whether it be from faulty liver metabo-

lism accentuated by excessive putrefactive bacteria, or due to local injury of the skin from scratching. Histamine, as is well known, is the cause for many reactions, such as swelling, intense itching, and even severe shock. Therefore, any disorder which might contribute to an excess of histidine, which is converted into histamine, must be considered.

Summary

Following is an outline of possible causes of pruritis ani:

I. Seven local or primary etiologies

1. Pathologic tissues (cryptitis, hemorrhoids, fissures, condylomata, fistulas, and skin tabs)
2. Internal parasites (Oxyuris vermicularis, Ascaris lumbricoides, Taenia saginata, and Trichomonas rectalis)
3. External parasites (Trichomonas vaginalis, scabies, and Pediculus corporis or pubis)
4. Mycotic infection (Monilia and the yeastlike fungi, Cryptococcus or Torulopsis, Candida albicans and immitis, epidermophytosis, and trichophytic fungi)
5. Allergy (idiosyncrasies and food reactions, "gormandizing" and high food seasoning, and drug reactions, as to cocaine, quinine, arsenic, and dermatitis venenata)
6. Altered biochemistry (drug addiction and alcoholism, high pH of intestinal flora, histamine, intestinal toxemia, indole and skatole, Streptococcus faecalis, and Escherichia coli)
7. Itch-scratch syndrome (uncleanliness, perianal skin hypertrophy due to trauma, psoriasis, histamine, and neurodermatitis)

II. Seven remote or contributing etiologies

1. Remote infections (bronchiectasis, pyorrhea alveolaris, Escherichia coli, Streptococcus faecalis, Bacillus coli communis, and gram-positive Diplococcus and Staphylococcus)
2. Blood dyscrasia (lithemia, pernicious or secondary anemia, increased bile salts, and cholesterol imbalance)
3. Hormone and endocrine imbalance (hypopituitarism, thyroid dysfunction and hypoadrenia, estrogenic and androgenic imbalance, senile and menopausal influence, and urticaria subcutanea)
4. Systemic disease (achlorhydria, atony, Hodgkin's disease, diabetes, gout, and organic reflexes such as prostatitis and cystitis, uterine and ovarian tumors and cysts, urethral stricture, and phimosis)
5. Vitamin deficiencies (avitaminosis, a dietary deficiency of vitamins, especially B complex, A, D, and C)
6. Psychosomatic factors (worry, fright, overwork, sedentary occupation, heterosuggestion and autosuggestion, and associated maladies such as melancholia, psychosis, and repressed sexual urge)
7. Altered liver metabolism (cholestasis and cirrhosis, intrahepatic and extrahepatic obstruction, regurgitation of biliary acids, degeneration, and necrosis).

The proctologist must recognize that the pruritus ani patient is in a serious condition and that he must be alerted to the facts; that his itching may be a symptom of one or more of several causative factors; that he cannot properly be treated until the cause is known; and that the three most likely backgrounds for his condition are parasites, pathologic intestinal flora, and faulty biochemistry.

The opinions of various noted authorities on this subject have been cited. All have proved their theories on certain percentages of cases treated; yet no one regimen can be conclusive since no single therapy is applicable to all.

It must be realized that, while pruritus ani is not strictly a protologic problem, the symptom does arise in this specialty field, and proctologists must assume responsibility for apprehending the cause and render service until a satisfactory termination of each case is accomplished.

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Dynamic concept of psychopathology

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Since science has become aware of the presence of phenomena of solely psychic origin, both normal and abnormal, it has attempted to explain these phenomena by the means employed in solving the problems of somatic pathology. This has failed, to a great extent. This failure has been attributed, in part, to the lack of knowledge concerning the physiology of the nervous system. To compensate for this deficit in neurophysiologic understanding, investigators at the end of the nineteenth and early in the twentieth century attempted to cross this frontier by the utilization of a new modality. Thus, psychoanalysis was born.

Prior to this time, psychiatry was analogous to the practice of symptomatic medicine. Its literature was almost wholly of a descriptive nature. Psychoanalysis has made it possible to trace the pathogenesis of mental disease. The symptoms of the sick mind are no longer the only considerations, but the sick mind itself is allowed to speak and thereby bring forth the origins of its pathologic conditions. The patient retraces the course of his illness to its onset. This is not too unlike the anamnesis of somatic disease, except that the psyche reveals itself begrudgingly and many detours and pitfalls are encountered.

Slowly, the pioneers in psychoanalysis began to learn how to guide the patient in his quest for understanding. This understanding, in its full emotional sense, has been found to be tantamount to recovery. The evaluation of these recollections revealed the essence of the psychopathology and led to the formulation of the psychoanalytic theory of human behavior.

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In the psychologic science as in the physical sciences, man has postulated what he could not prove and conceived what he could not see. Just as we had an atomic theory long before there was an electron microscope to see molecular structures, today we have the concepts of ego and id without knowing what areas of the body, if any, they belong to. As mysterious small particles are used to change atomic configuration by bombardment, so we use tranquilizers, antidepressants, and shock therapy with no clear knowledge of their physiologic action in relation to the psychologic changes they may produce.

Modern psychiatry is a blended science. It is founded in the tradition of medical observation and description, with attempts at physiologic and biochemical explanation, and permeated by psychoanalytic thought. The psychoanalytic theory, which was born through the formulation of the histories of thousands of patients into a *modus operandi* of human behavior, is dynamic—dynamic in the sense that it focuses attention on what has happened in relation to what is happening; that is, cause and effect.

The intent of this paper is to acquaint the reader with the change in approach, thought, and technic that a physician must experience in becoming a psychotherapist. I believe it can only be assayed at the point where both ways of looking at the situation are fresh in the mind. It is hoped that a clearer understanding of the dynamic approach in psychotherapy may be fostered herewith.

Approach to the patient

Unfortunately, the attitude, "Ah, just another neurotic," is quite common among physicians. Yet, this may be just as disastrous as the feeling, "Ah, just another C.V.A.," or, "Ah, just another diabetic." It is almost superfluous to point out the sizable percentage of suicides among neglected depressive persons. This type of thinking is self-defeating.

A most important aspect in the therapeutic situation is how the therapist relates to the patient. His impartiality and nonjudging manner are of prime importance, no matter how foreign are the circumstances unfolded. Many of these problems are usually solved in the analyst's personal analysis and others are overcome with experience. The patient should be treated as a guest one receives in his own home, who is enabled to feel welcome at all times.

Transference

Aside from the still existent "bugaboo" about psychiatry, at the beginning of therapy the patient feels that he is simply going to another physician. But, as the therapy proceeds, the relationship changes. These changes may be arbitrarily classified into three stages: (1) authoritative-receptive; (2) co-explorative; and (3) co-reactive.

These stages represent different degrees of libidinal cathexis (emotional charge) invested in the therapist. This patient-to-doctor relationship is called the transference. The doctor's feelings projected onto the patient are collectively termed the countertransference.

In psychotherapy, where the patient is seen generally once or twice a week and sits facing the therapist, the authoritative-receptive attitude gradually changes into the co-explorative phase. The relationship with the therapist acquires meaning for itself. The therapist is regarded with fond feelings (positive transference) or with hostile feelings (negative transference). In this type of treatment, the very essence of the psychopathology is rarely revealed directly, but insight is gained on a higher, yet parallel, level of development. The patient usually does not gain insight into infantile conflicts but does gain realization of the significance of the situations that came about due to these conflicts. The co-reactive stage of the transference is rarely achieved.

The relationship of the patient to the therapist in the sessions is a reflection of his relation to the rest of his world. This is the value of the transference. The patient basically relates to all human beings in a pattern which is in keeping with his neurotic outlook. The psychotherapist is incorporated into his environment and treated as part of it. This patient-to-doctor relationship is then simply an interhuman experience. Some of this is consciously shared, but a great deal occurs unconsciously. By making the patient aware of the occurrences, as they appear in his new relationship and by comparing them to other reality factors, the neurotic conflict unfolds and is then treatable.

In psychoanalysis, the patient is seen three, four, or even five times a week. During these sessions he preferably lies on a couch. The sole purpose of this is to allow relaxation and facilitate free association, which is simply the pouring forth of successive thoughts unabated. The patient chosen for analysis is usually, of necessity, from a higher socioeconomic level and therefore most likely has had the benefit of a higher education than the usual psychotherapeutic patient. Yet, each case must be given individual consideration in choosing a mode of treatment. The element of unreality connected with the analytic situation rarely allows psychotic patients to be put on the couch. More direct methods of analysis are utilized with psychotic patients in institutions.

In psychoanalysis, the co-reactive phase of the transference is reached and used. In this phase, the analyst is not accepted merely as a confidant and friend, but is reacted toward as representing a figure of the individual's infantile family environment: father, mother, brother, sister, or a representative surrogate. The relationship thereby fostered is similar to one that existed in his childhood psychic or external reality, and thus the neurotic conflict unfolds itself. In the analytic situation, often the basic, lowest levels of the conflict are encountered and many memories previously repressed again become conscious. Some of the veil occluding recall of the first 4 or 5 (or more) years of life is lifted, allowing insight. Naturally, in conditions where the discovery of this material would be too threatening, analysis would not be the best choice of therapy. In the case of many perverts, this is so. Therefore, each patient must be evaluated individually as to the type of therapy suitable for his case.

The unconscious

The work of the psychotherapist is to guide the patient to the basis of his problems simply by listening to him—listening not always to the words but to the meaning behind the words, for these reveal the unconscious wishes and fears. Learning the language of the unconscious occupied many early psychoanalytic workers through the course of thousands of analyses, and this represents a great part of the acquisition of a therapist in training. This knowledge is then utilized to interpret the language of the patient, in view of the facts known about him and his life situation.

The following example may clarify what is meant by the language of the unconscious: A child therapist engages in play therapy with a 5-year-old patient. The patient states emphatically, "I love my brother." But, just a few seconds earlier, the therapist had seen this very child poking and kicking his 3-year-old brother and tormenting him in as many ways as he could. What the child actually wants to say is, "I hate my brother," but he has been forced by his interpretation of his parental environment to express love for his younger rival. There is sibling rivalry for parental affection, and the older child fears to express hostility openly toward his brother. He fears being punished and losing that affection, some of which has already been usurped by the young intruder. Therefore, he says with emphasis, "I love my brother." The child is utilizing reaction-formation (opposite thought and behavior from that which is actually desired).

This is a defense mechanism, a subject which will be referred to again in this paper. This is what is meant by the language of the unconscious; the therapist must interpret it for the patient so that he will gain understanding.

Interpretations

The interpretations may be grouped simply into three types:

1. Comparisons, of various aspects, to facilitate the recognition of the relationship of different associations.
2. Clarifications, to promote insight.
3. Conflict (neurotic) evaluations, wherein direct aspects of the basic pathologic condition are interpreted.

The first two groups of interpretations are utilized almost throughout the therapy, whereas the third group is understandably reserved for later periods only. It is only with experience that a proper sense of timing is acquired. Interpretations should not be made until the patient is ready for them. Classically, it is recommended that an interpretation be made only when it is already "on the tip of the patient's tongue." Incorrect timing results in the patient and therapist just having a discussion, and the interpretation is meaningless. It may be rejected or, even if it is accepted, it is done so intellectually and not emotionally.

Stages of therapy

The therapeutic situation itself is usually divided into three stages:

1. Early phase, which includes the initial interview and the history taking.
2. Middle phase, which commences with the first interpretation.
3. End phase, in which consideration for the termination of therapy is begun and carried through.

These stages often overlap and interpretations may be made, not only during the history-taking, but occasionally in the initial interview as well.

Neurosis

It is not within the intended scope of this paper to discuss individual mental diseases, but a general outline may clarify at least some semantic difficulties.

All functional diseases of the nervous system may be termed neuroses. These are to be separated from organic lesions of the nervous system. The neuroses present symptomatology ranging from the most minute deviation from the norm to the most insane and bizarre behavior. The true functional psychoses, which at this writing still have no proved organic basis, are a special group which may be quite separate from the other neuroses or merely a severe exaggeration of the milder conditions.

The neuroses may be further subdivided into:

1. Psychoneuroses, including phobias, anxiety reactions, depressive states, obsessive-compulsive states, and conversion hysteria.
2. Organ neuroses, which are commonly called psychosomatic (psychophysiologic) conditions.
3. Character neuroses, which include sociopathic (psychopathic) personalities and disorders of character structure.

Neurosis represents conflict—the conflict between what the individual, as a member of the animal kingdom and recipient of its heritage, would like to do and what he is confined to doing by the laws of society. In all the neuroses, save the last mentioned, the conflict is within the patient and is manifest in the symptoms which represent a compromise. The sociopath has externalized his conflict, since his behavior is acceptable to himself (ego-syntonic) and he gratifies his conflicts in antisocial behavior. The conflict in psychosomatic disease is expressed by anatomic damage in the form of an ulcer crater, bronchial spasm, dermatitis, hypertension, and so forth. Psychosomatic disease differs from conversion hysteria in that the insult is permeated via the autonomic nervous system rather than affecting the musculoskeletal network. Dynamically, the difference is much greater. Whereas in patients with peptic ulcer and psychogenic asthma we would attempt to explore aspects of the dependency traits, in conversion hysteria we would attempt to lift repression.

The reservoir of atavistic impulses representing one of the combatants in the conflict is called the id. That aspect of the psyche which mediates with the outside world, the ego, throws up defenses against the id to keep its threatening desires from becoming manifest. These ego defenses were discovered and studied by listening to and observing thousands of patients. They can be detected by the trained ear, as reaction formation was detected in the case of the 5-year-old boy mentioned earlier. Later on in the course of therapy, these defenses are exchanged or eliminated as the patient begins to master the conflict. Early in therapy these defenses have diagnostic significance.

Diagnosis

As is true with all branches of medicine, psychiatry gives the satisfaction of discovering within the patient the signs and symptoms of the disease process, enabling the practitioner to make a diagnosis. But, again, the diagnosis is often based on the dynamic aspects and not merely on manifest symptomatology.

An illustration of a dynamic psychiatric diagnosis is as follows: In the popular descriptive sense, we may think of an obsessive-compulsive neurotic as an individual who is ritualistic and performs certain acts over and over again. But this is the compromise manifestation of only one of the ego defenses involved, namely undoing (the attempted dissolution of an undesirable situation by repetitive activity). In obsessive-compulsive neurosis, besides undoing, reaction formation, isolation, and regression are

utilized by the ego to combat the id. Not every obsessive-compulsive neurotic utilizes the defense of undoing to the extent that manifest ritualistic symptoms are detectable. This is especially true early in the neurosis, but the trained ear can detect the employment of isolation (separation of the emotion from the fact) and reaction formation as has been shown. Therefore, the diagnosis of obsessive-compulsive neurosis can be made correctly in patients not exhibiting characteristic rituality.

In other words, dynamic psychiatry judges the fight not by the bruises acquired, but rather by the types of punches thrown. It gets inside and evaluates the battle and does not merely stand off and admire the scars.

Technic

The patient, in the therapeutic sessions, is guided to explore as much of his conscious life, dream life, and phantasies as possible. He speaks freely of those things he considers important and is encouraged not to cast anything off as being insignificant. He is to express all that occurs to him. At times this becomes painful and threatening, and the patient erects defenses originating within his own psyche which prevent material from becoming conscious to him. The defenses are maintained by resistances felt toward the therapist. As these are recognized, they are pointed out to him; only with his understanding and acceptance of them are they overcome. Through free association, material previously unconscious is freed from the bonds of the unknown and, via the propellent of attention, these memories pass through the preconscious and become conscious. The regaining of this repressed material aids the patient in achieving insight into his problems.

The adult is in reality a grown child, as the tree is a product of its roots, and the years of early development are the ones which greatly determine the course of life. It is well known that during the first trimester of pregancy a viral infection such as measles may cause the infant to be born with a congenital defect. Yet this same disease inflicted upon the mother-to-be later in her pregancy will not harm the infant. The reason for this is that it is only during the first trimester that actual differentiation of organ systems takes place. After they have developed, they merely grow in size, and then no developmental defect can occur. The same is true with abnormal psychology. Trauma, insignificant to the psychologically mature individual, when inflicted upon the developing psyche, may cause severe damage apparent only years later.

It is the aim of psychotherapy to explore and focus attention on the traumatized areas of the developing psyche and the reactions and adjustments to them. The individual may then reroute his mode of functioning and be able to live a reasonably normal life. This is not too unlike an operation to ligate the patent ductus arteriosus, which is often present in a child born to a mother who had measles in the first trimester of pregnancy. While the process is of an entirely different and more lengthy nature, the results are nonetheless just as dramatic.

The success of psychotherapy does not depend upon the merit of the interpretations made to the patient, but in the ability to guide the patient toward making his own interpretations. Often, the interpretations are construed by the patient as being analogous to the criticism of others, and they are then of questionable value. In order to gain independence and maturity, the patient must learn from his own relationship to the therapist, and not merely absorb what is said to him. This learning may take place on an intellectual or emotional level, and either can be of benefit to the patient.

The reasoning utilized by the therapist during psychotherapy differs greatly from that which he acquired during his medical training. In the medical situation, the physician seeks the etiologic basis of the disease and outlines the therapy. The patient is the passive recipient. In psychotherapy, the patient relates the etiologic factors in an active manner. It is the therapist who is the passive recipient. Therefore, the psychotherapist must unlearn behavior which has become part of him during his medical training and previous years of medical practice.

Summary

This paper has been presented to illustrate the highlights of the transition of thought and technic which occur in the study of psychiatry. Throughout, the dynamic and psychoanalytic concepts have been stressed. It is hoped that some of the so-called mysteries of what goes on in the psychiatric ivory tower and upon the couch have been clarified.

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A report to the membership (continued)

TRUE B. EVELETH, D.O., Executive Director, American Osteopathic Association

During the past year, much of the attention of the Board of Trustees and the Central Office staff, as well as many others, has been directed toward the unfortunate capitulation of osteopathy in California. The House of Delegates was called in special session last January. The California defection has been costly to the profession from many standpoints and the damages still mount.

The proposed liquidation of osteopathy by joint action of the California Medical Association and the California Osteopathic Association has progressed from the planning stage through the signing and ratification of a pact, the enactment of enabling legislation, and the agreement to surrender the osteopathic college. These developments have been reported in detail in the January, March, April, and June issues of THE JOURNAL.

Still pending are court action or actions and the vote of the people of California, either of which could stop, or delay, the elimination of osteopathy in that state.

In April, the newly chartered divisional society, Osteopathic Physicians and Surgeons of California, filed a complaint in the Superior Court of the State of California in Los Angeles County, protesting many facets of the C.O.A.-C.M.A. agreement. This action amounted to a plea for permanent injunction to stop the merger on the grounds that is was illegal. At the same time, a plea for temporary injunction was filed to stop further activities toward fulfilling the merger agreement prior to the trial on the complaint. The plaintiff, O.P.S.C., contended that irreparable damage would result from such activities. This petition was denied by the judge's decision that irreparable damage would not necessarily occur and that, if actions were taken which would cause such damage, the court would consider the petition.

Now pending is the trial on the complaint and the petition for permanent injunction. In this trial, the issues are on the legality of the several provisions of the signed agreements to merge. The defendants, C.O.A. and C.M.A., filed demurrers to the complaint on June 6, admitting, in a sense, the facts contained in the complaint, but denying that they were illegal. The court hearing on the demurrer is scheduled for July 17, 1961. The decisions rendered then will establish the opinion of that court as to the legality of each of the charges in the complaint. Actual trial proceedings on the complaint would then be scheduled several months later. The opinions rendered on the demurrers would influence the preparation for the pleadings at the subsequent trial.

Whether or not the court will grant relief by forbidding further implementation of all or part of the agreement cannot be foretold. Such issues are not black or white, and court decisions are usually appealed by the losers until the Supreme Court rules with finality.

The basic problem facing this profession in California is not a legal one and cannot be resolved by the courts. A significant number of D.O.'s are intent on acquiring M.D. degrees and are willing to ac-

cede to the terms dictated by a power which is solely interested in elimination of osteopathy and the acquisition of the Los Angeles College. While the courts may find against the manner in which the parties have agreed to consummate their contract, it will not alter the interests of the defendants nor prevent their continued search for a means to the desired end.

In order to create an atmosphere in which such a deal could be made, many real and important values had to be destroyed. The value of osteopathy itself, the quality of osteopathic education, the sincerity of leaders within the profession, and many other factors had to be distorted. This has been done and little is left of matters osteopathic in fact or in the minds of Californians.

Several resolutions have been presented for Board and House consideration with the purpose of placing safeguards in the By-Laws which would prevent an osteopathic college or divisional society from defecting from the objectives or policies of the A.O.A. Whether such action is wise poses one question to be considered. It must be borne in mind that no amendment to the A.O.A. By-Laws would affect or alter the autonomy of a college unless the by-laws or charter of the college included like provisions. The ability of this profession to protect itself against the outside forces now massed to destroy it depends upon whatever unity, determination, and conviction exist within the profession. During a seige, the defenders must display an extraordinary brand of determination and conviction which more than matches that of the attacking forces. Let there be no mistake about the determination of the attacking forces. The C.O.A., which has maintained consistently that the other states would follow the California plan and has contended that many states were ready to secede, is propagandizing the nation and offering M.D. degrees and traveling counsel to anybody anywhere. The C.M.A. has repeatedly said that the American Medical Association is in full accord, and A.M.A. leaders have professed that other states would follow the California pattern.

A nucleus of D.O.'s with principles and courage have established a beachhead for the purpose of preserving the rights of the public, the continuation of osteopathy as a profession, and a place for those who wish to remain osteopathic physicians and surgeons. Doubtless there are hundreds of others with like principles who lack the courage to be so counted. Many factors create a climate of apprehension which is difficult for free Americans to comprehend. This has been brought about by a combination of ruthlessness, craftiness, distortion of facts, and downright falsehoods, backed by Machiavellian justification of unscrupulous means to gain a desired end, beyond the comprehension of most practicing physicians.

The devastation is great and in many areas irreparable. Our attention to the matter comes late since the secret negotiations were uncovered a short 2 years ago. The A.O.A. is severly criticized both for interfering in the affairs of California and for not preventing the disaster. Recriminations are natural and inevitable, although useless and destructive. Recrimination is the easiest way to participate in a serious problem, but usually it is the only contribution made by those who resort to it.

Osteopathy as a school of medicine has, through its national and state organizations, declared its determination to continue its existence. The House of Delegates of the American Osteopathic Association has directed the Board of Trustees "to continue to use any and all means possible to assure the continuation and development of the osteopathic profession and its educational institutions in the State of California in the public interest." The Board has conscientiously attempted to carry out that assignment. Assistance has been given the Board by many individuals; considerable money has been expended, and more is committed in the interest of salvaging everything possible. Some D.O.'s in California have spent more time and money than they could afford, but too few of their colleagues have backed them or even encouraged their efforts.

At this moment when every eye should be focused on our objectives, there is no time for selfish indulgences. We must realize that our preferences have to be weighed against the problem we are trying to solve or they will become a part of the problem. I plead for closer, more harmonious attention to the purposes for which we are organized. The forces determined to destroy osteopathy have extended beyond the borders of California; they have invaded many states. These are organized forces encouraged by the apparent successes in California and by learning that destruction of osteopathy can be accomplished through conspiracy with disloyal members.

The osteopathic profession's determination to defend itself against destructive forces means active defense wherein all its segments, schools, specialty colleges, and affiliate bodies, as well as individual members, merge in the common interest. All must realize that to pursue individual interests dilutes the united front and thus the effectiveness of the defense. Associations such as the A.O.A. do not operate with the efficiency of industry. Voluntary service, regardless of its dedication, enjoys an independence that would ruin a business corporation very quickly. No bulletin of a branch office of the Standard Oil Company constantly contends that the Standard Oil Company is stupid and that its directives should be ignored. No such bulletin cheers another branch office for pushing a competitive line. A voluntary association is dependent upon the determination of its membership to exist and further the purposes for which it was organized. Without this, no association can withstand the stress of an organized attack. If the osteopathic colleges, the divisional societies, and the other groups related to A.O.A. will not unite in a common front, the A.O.A. cannot protect the profession.

Despite its current problems, if this profession chooses, we can be entering the greatest era in

the history of osteopathy. There is concrete evidence that philanthropy will support osteopathic education primarily because of its distinctive contribution. During these difficult days, we are encouraged by friends we did not know we had and by a resurgence of interest within the profession in the principles of osteopathy and the development of the profession. It is in this direction that we must now exert our efforts for it will be through these means that this profession will reach the zenith of its own development and of its service to mankind.

Executives' health

The executive has six particular occupational hazards, according to Dr. Lemuel C. McGee, of Wilmington, Delaware, a speaker at the recent meeting of the American College of Physicians, in Miami Beach. These hazards are the cocktail hour, the banquet circuit of excess food and boring after-dinner speeches, the swivel chair, the need for decision making, the stress of dealing with people, and pleasurable vices.

Dr. McGee, who is cited by Dr. Howard A. Rusk in the May 14 issue of *The New York Times*, has a number of antidotes to offer. He recommends proper medical care and promotion of active resistance, both natural and acquired, to disease; adequate nutrition; periodic dental services; and exercise appropriate to one's age, sex, physical condition, and skills.

Also recommended were satisfying creative work and a sense of usefulness, wholesome play and recreation, a salubrious living and working environment, ingenuity in avoiding frustration, a supporting philosophy of life, and suitable rest and relaxation.

Periodic health examinations were emphasized by another speaker at the conference, Dr. James P. Baker, medical director of the Greenbrier Clinic in White Sulphur Springs, Virginia. The executives' program of his clinic includes health education, early detection of disease, and reassurance.

In one series of 5,000 executives examined at the Greenbrier Clinic, 467 (8.5 per cent) were found to have colonic polyps. There was malignant change in 40 cases. Immediate operation was performed, and all but 2 individuals are still living and apparently cured.

To the regular examinations carried out at this clinic has been added a test for ocular tension. Of 2,500 persons routinely examined by this test, 240 had unsuspected borderline or elevated values. All were referred to ophthalmologists, and twenty four were found to have early glaucoma.

At another clinic, the Silver Hill Foundation in New Canaan, Connecticut, an "emotional check-up" has been added to the periodic health examination for executives. Psychologic testing is used regularly, and the patient is given opportunity to discuss problems of tension, personality, family, and business.

According to Dr. William B. Terhune, medical director of the Foundation, nearly all who have participated in this psychologic testing program have reported increased insight into problems of which they had not been specifically aware, but which had been manifested in increasing irritability, tension, and lack of efficiency.

The Times also reports that a participant in the meeting quoted some medical advice from an unusual source: Leroy (Satchel) Paige, the ageless baseball pitcher, has this prescription for good health:

> Avoid fried foods which anger the blood. If your stomach disputes you, lie down and pacify it with cooling thoughts. Keep the juice flowing by jangling around gently as you move. Go very light on the vices such as carrying on in society—the social ramble ain't restful. Avoid running at all times. Don't look back. Something may be gaining on you.

Prevention at its best

Preventive medicine at its best lies in the suggestion of Assistant Surgeon General A. L. Chapman that physicians include in their examinations of patients an assessment of competence in the driving of automobiles.

In a guest editorial in a recent issue of *Medical Tribune*, Dr. Chapman, Chief of the Division of Accident Prevention of the United States Public Health Service, advances the proposition that it is as logical for a physician to investigate a patient's physical and mental capacity to handle an automobile as it is for him to advise what and how much a patient may eat and drink, and what exercise he may take. From the viewpoint of society, it is far more important. An incompetent or irresponsible driver jeopardizes more lives than his own.

It is not difficult to accept Dr. Chapman's reasoning. A physician is in the best possible position to discuss with a patient anything pertaining to health. He has a better than even chance of being listened to. Face-to-face consultation precludes blaming the "other fellow," to whom newspaper and billboard admonitions are usually considered to apply.

As a part of his advice to promote a patient's well-being, a doctor can prescribe the use of seat belts, padding, and crash-proof glass. He can also, especially with young patients, speak out on the subject of social responsibility on the highways.

Dr. Chapman's suggestions merit action by every practicing physician. They add a powerful weapon in the battle doctors have long fought against automobile accidents and fatalities. The examination of particular patients on their ability to drive can add impact to the service physicians have for many years given in safety councils; in medical testimony

in accident cases; in urging immediate, round-the-clock hospital emergency service; and in demanding in the face of public irresponsibility and industrial resistance, the incorporation of proved safety devices in automobile manufacture.

The Assistant Surgeon General also opens up a direct route to the public conscience, upon which the "epidemic of the highway," should rest with crushing weight. Today, in this country, every active person is threatened with incapacity or extinction through highway accident. The automobile takes more lives of persons between 5 and 24 years of age than any disease. Its fatalities every year deprive the nation of one and one-half million years of life expectancy.

Nor is it an ill that will abate. In the U.S. now there are 82,000,000 drivers of automobiles. By 1971, according to estimates, there will be 120,000,000. Accident prevention becomes a crucial need. A physician, as he examines a patient, can do much to meet it. Only one person can do more: the patient being examined.

The paradox of progress

Man has advanced as he has learned to make use of the world's resources. He was warmer and better fed when he learned to kindle a fire. He was more comfortable when he could leave the dampness of his cave for a shelter built of mud and stone. He could travel when he had fashioned sail and wheel.

Through the ages, man, curious and intrepid, has explored the earth and has turned its properties to his service. Within the present century, he has moved at an incredible pace. He has learned to build cities of steel and glass and to project his voice across vast distances; he has developed mechanisms to carry him at unbelievable speeds over land, under water, and through the air. For his immediate convenience he has fashioned a push-button environment. He has released the energy of the atom, and is now venturing beyond the earth he has for so long explored.

In this headlong evolution, man has turned his attention to the liberation of his own physical and mental powers. He uses the earth's resources to cure and strengthen his body. He knows the necessity for protection against bacteria, and uses of anesthesia and x-ray. He has developed and specialized his curative skills. He is exploring the powers of radiation. He knows how to lengthen his life, save his newborn, and make his years more productive.

Yet as man progresses, he trades one set of hazards for another. As he broadens and betters his living, he multiplies the force and extent of his dangers. This is perhaps the paradox of progress. It is also the challenge thrown at the feet of medicine. The physician, even while he cures the sick, works for general public health and converts today's discoveries to curative purposes, must also prepare himself to meet the exigencies of tomorrow.

The physician today moves among the horns of countless dilemmas. The people he cares for are better housed, betted fed, and more comfortable than any people of history. Yet environmental pollution may be exposing them to chronic poisoning. People have more opportunity to learn, more freedom, and greater resources, yet rising numbers of them break under the stress of living. They can look forward to longer life, but can annihilate themselves at the turn of a switch.

In this maze of paradoxes, there are countless wars to be won. Immediate among them is the battle against the contamination of air, water, and food that has come as a by-product of today's technology. As chemistry produces synthetics for man's convenience, as increasing thousands of automobiles multiply sources of smog, as industrial waste materials are dumped into streams that also supply drinking water, the health of all citizens is endangered. Again man increases his well-being, but exposes himself to new dangers.

Research estimates that 15 per cent of cancer may be traced to pollution of the air. Ten thousand communities are affected by smoke and fog. Synthetic chemicals that kill mosquitos, clean clothes, and wash pots and pans are being flushed as waste into rivers and lakes. Untreated human sewage is being dumped into streams. Four thousand chemicals, some of which may be harmful, are used in the processing, storing, and handling of foods.

Medicine joins with government and private health agencies in the fight against contamination of air, water, and food. It fights the "dread diseases," struggles with the mysteries of mental retardation, and reaches out to the people of emerging nations.

But this is not enough. Medicine cannot be content with facing the battles of today. It must attune itself for the world of tomorrow. Man, curious and intrepid, skirts the potencies of new and awesome forces he has released, and prepares himself to explore planets other than his own. Man has never had so much, nor placed it in greater jeopardy. The man of medicine must assume his role in this new and perhaps greatest of all paradoxes.

A.O.A.—1960-1961 943
Department of Public Affairs 946
Council on Federal Health Programs 947

A.O.A. ACTIVITIES

A.O.A.—1960-1961

We must never lose sight of the fact that progress comes through a day-by-day, year-by-year progression of steady labor, in big and little things.—True B. Eveleth, D.O., Executive Director, American Osteopathic Association.

▶ In the year-by-year story of the American Osteopathic Association, July is the month of accounting. It is then that the annual meetings of the House of Delegates and the Board of Trustees are held, and that the work of the year is reviewed.

Early this month, the House and Board met in the Drake Hotel in Chicago. Some fifty bureau and committee chairmen, A.O.A. administrative officers, and other members of Central Office staff reported on the work of the Association in 1960-61. Here, as a preview of the official reports that will be published in THE JOURNAL in months to come, are some of the year's developments and events:

1960-61 appointments • A Bureau of Disaster Medical Care was created by the Board of Trustees last summer in Kansas City. Dr. Raymond W. Hanna, Independence, Missouri, was made Bureau chairman, and ten regional chairmen were subsequently named. Some 150 physicians and hospital administrators attended a Bureau-sponsored seminar, held in Kansas City in April.

The year's work of the Committee on Medical Care Plans, under Chairman Theodore F. Classen, Warrensville Heights, Ohio, reached its height in the Conference on Health Insurance and Medical Care Plans which immediately preceded this month's meeting of the House of Delegates in the Drake Hotel in Chicago.

NHC directorship • At the March meeting of the National Health Council in New York City, Dr. George W. Northup, Livingston, New Jersey, was named to the Council's 35-member board of directors.

Dr. Roy J. Harvey, 1960-61 President of the American Osteopathic Association.

An A.O.A. Committee on Health Care of the Aging was appointed, with Dr. Vernon H. Casner, Kirksville, Missouri, as chairman. Ten physicians represented the profession at the 1960 White House Conference on Aging.

In Atlanta, Georgia, in January, Dr. Earl E. Congdon, Flint, Michigan, took his place as a member of the Advisory Committee on Poliomyelitis Control, an agency of the Department of Health, Education, and Welfare of the United States Public Health Service. Dr. Hanna and Dr. Carl E. Morrison, St. Cloud, Minnesota, were named as consultants to the USPHS Division of Health Mobilization.

A.O.A. President Roy J. Harvey, Midland, Michigan, was one of ten civilians to be chosen from the disciplines of medicine, law, and law enforcement to attend the third Armed Forces Forensic Sciences Symposium held at the Armed Forces Institute of Pathology in Washington, D.C. in May.

Film on osteopathy • "American Doctor," a film on the founding and development of osteopathy, produced by the A.O.A. Division of Public and Professional Service, was completed. Shown first at the

A.O.A. annual convention, ninety-eight prints are now being used throughout the country for television showings and for use at such meetings as those of service clubs, women's clubs, and vocational guidance groups.

A new vocational guidance manual, "Opportunities in Osteopathy," was published during the year. The author is Lawrence W. Mills, director of the A.O.A. Office of Education. "Focus on Research," the fourth in the series of "Focus" booklets, was produced, with Leonard Heffel of the Division of P. and P.S. as author. "Focus on Education" was reprinted. The *AOA News Bulletin*, published monthly, continued to reach a selected public, as well as the members of A.O.A.

A.O.A. staff members at Miami Beach convention—Luise Van Hook, administrative assistant, and Michael L. Roche, service department, discuss mimeographing methods.

Educational revisions • Fundamental revisions in osteopathic educational processes, instituted in 1959, became operative. The changes clarify the authority and function of committees making up the A.O.A. Bureau of Professional Education, and call for "across the board" upgrading in teaching osteopathic principles and technic.

During May, seminars on medical writing, developed and conducted by the A.O.A. Editorial Department, were instituted in five osteopathic colleges.

The Bureau of Research, in a departure from custom, met this year at the Kirksville College of Osteopathy and Surgery, Kirksville, Missouri, rather than at Central Office in Chicago. Thirteen research programs were reviewed. The fifth annual Conference on Research was held in Central office in March.

Midwinter convention • In a

Ohio delegation with A.O.A. President-Elect at Miami Beach convention—Left to right, seated, Drs. A. C. Fuller, Jack M. Wright, Jack D. Hutchison, and Leonard D. Sells, 1960-61 president of the Ohio association; standing, Drs. James E. Walker, President-Elect Charles L. Naylor, John W. Hayes, Carl P. Gephart, and Donald J. Ulrich.

break with tradition, the Association held its Annual Convention in midwinter, in the resort atmosphere of Miami Beach, Florida. The annual meeting of the House of Delegates, however, was held in July. This separation of the didactic and legislative is pointed toward a single large meeting of all osteopathic clinical groups.

Professional and public support of osteopathic education and institutions continued on the rise. Professional contributions alone carried the Osteopathic Progress Fund, close to a million dollars for the year. The Student Loan Fund rounded out 30 years of service by totaling $125,300 in this year's loans to osteopathic college students. The Christmas Seal Campaign exceeded $70,000 in returns, to be used for student loans and research. Educational grants totaled $48,000.

The number of beds in registered and approved hospitals this year totals 11,730, an increase of 1,075 over last year. The number of applications for entrance into osteopathic colleges for the coming year is the highest since 1951. The combined gross income of the Association from advertising and supplementary services is the highest in Association history.

Thus goes a sampling of nationwide activity of the A.O.A. in the year 1960-61. The recital is far from complete. It is not material for newspaper headlines. But it represents the kind of year-by-year work that has for 65 years marked the development of the osteopathic profession.

Above, scenes from "American Doctor," film on osteopathy produced this year by the American Osteopathic Association. Top, Dr. Still (Donald Woods) meets "cold front" of established medicine. Below, Dr. William Smith (Lewis Martin), early supporter of the new concept, accompanies Dr. Still on his rounds.

Left—Government display on water pollution, a featured exhibit at the Miami Beach Convention, held in January.

Association takes out membership in ANCHEP

▶ Organized participation in the work of world-wide health education has been opened for the American Osteopathic Association in its recently acquired membership in the American National Council for Health Education of the Public and, through it, in the International Union for Health Education.

Three A.O.A. delegates • The Association membership was announced at the Third Annual Delegates Assembly of ANCHEP, held in April at the New York Academy of Medicine, New York City. President Roy J. Harvey, Midland, Michigan, and A.O.A. Executive Director True B. Eveleth, Chicago, were named as delegates. At the same meeting, Dr. Robert D. Anderson, Philadelphia, A.O.A. trustee and chairman of the Bureau of Public and Industrial Health, was appointed to the Membership Development Committee. Dr. Anderson has for some time held an individual membership in ANCHEP, and is also a delegate.

IUHE and ANCHEP • The International Union for Health Education, of which ANCHEP is the American representative, is a world-wide organization working to promote health through education. Working with the World Health Organization, it provides an international clearinghouse for the exchange of information, and promotes research and the development of technics in health education. It was founded in Paris in 1951. Prof. Giovanni Canaperia, of Italy, is now its president, and Clair E. Turner, Dr.P.H., Arlington, Massachusetts, is its chief advisor.

The A.O.A. brings to thirteen the number of voluntary and professional societies that make up ANCHEP's active membership. Its president is Henry van Zile Hyde, M.D., of the United States Public Health Service, and Howard W. Ennes, Jr., New York City, is its secretary. In addition to individual and active memberships, ANCHEP's organization includes associate, affiliated, and sustaining memberships.

International meeting in 1962 • ANCHEP will be host to the 1962 International Conference on Health and Health Education, to be held in Philadelphia, June 30 to July 7. This will be the fifth such conference, and will be co-sponsored by the World Health Organization.

Reprints of "Focus on Osteopathic Education" are available from Order Department 212 E. Ohio St., Chicago

Peter G. Meek succeeds Phil Ryan as NHC director

▶ Peter G. Meek, Ridgewood, New Jersey, takes office as executive director of the National Health Council on August 1. He succeeds Philip E. Ryan.

Mr. Meek's appointment was recently announced by NHC President James H. Sterner, M.D., Rochester, New York. He comes to the Council from his post as director and northeast regional representative for the National Society for Crippled Children and Adults.

Dean Dranias joins staff; Robert Bradner resigns

▶ Dean Dranias, a native of Chicago, has become a member of the staff of the A.O.A. Division of Public Health and Professional Service, as announced by A.O.A. Executive Director True B. Eveleth.

Mr. Dranias is a graduate of Northwestern University Medill School of Journalism, and majored in radio and television work. He formerly was employed by the National Safety Council.

Robert Bradner, a member of P. and P.S. staff for 2 years, resigned early this month to become news editor of *Savings and Loan News*, a national trade publication produced in Chicago.

DEPARTMENT OF PUBLIC AFFAIRS

Bureau of Public Education on Health

Indiana legislature passes new county hospital bill

▶ Soon to go into effect in Indiana is Senate Bill 436, which was enacted into law during the 1961 Indiana general assembly. Titled the New County Hospital Non-discrimination Law, it provides that any hospital supported by county funds may be used by all taxpayers and their dependents within its jurisdiction. It further specifies that the people may choose their physicians, provided the physicians hold unlimited licenses issued by the Indiana State Board. The full text of the new law follows:

Any hospital supported by funds provided by a county unit of government authorized to levy taxes shall provide the use of its facilities to all taxpayers within its jurisdiction and to dependents of such taxpayers under the care of the physician of their choice if such physician possesses a license to practice medicine without limitations issued by the Indiana State Board of Medical Registration and Examination: Provided, That the provisions of this act shall not deprive the governing board of such a hospital of the right to adopt and enforce reasonable rules and regulations concerning the use of such hospitals and its facilities by such physicians: Provided, further, That denial of the use of such hospital and its facilities by a licensed physician who is a resident of the county shall be prima facie evidence of a violation of this act.

Bills in Congress

▶ H.R. 1 Mr. Anfuso of New York. Establishes under the National Science Foundation a National Science Center consisting of a National Science Academy and research institutes in meteorology, oceanography, medicine, and such other research institutes as the Director may deem necessary.

H.R. 10 Mr. Keogh of New York. To encourage the establishment of voluntary pension plans by self-employed individuals. Amends Internal Revenue Code to authorize qualified self-employed individuals an income tax deduction of up to 10 per cent or $2,500, whichever is smaller, of self-employment earnings. Passed House June 5, 1961.

H.R. 27 Mr. Fogarty of Rhode Island. Authorizes a 10-year program of grants for the construction of medical, osteopathic, dental, and public health educational facilities.

H.R. 67 Mr. Byrnes of Wisconsin. To repeal the maximum limitations which apply for purposes of the income tax to the deduction for medical care expenses.

H.R. 86 Mr. Frelinghuysen of New Jersey. Permits a 30 per cent credit against the individual income tax for amounts paid as tuition or fees to certain public and private institutions of higher education.

H.R. 94 Mr. Holland of Pennsylvania. To amend the Social Security Act to provide hospitalization, nursing home, and surgical services for OASDI beneficiaries. Same as Forand bill of previous Congress.

H.R. 133 Mr. Roberts of Alabama. Establishes a National Accident Prevention Center in the Public Health Serivce.

H.R. 134 Mr. Roberts of Alabama. To provide that seat belts sold or shipped in interstate commerce for use in motor vehicles shall meet certain safety standards to be prescribed by the Secretary of Commerce.

H.R. 185 Mr. Walter of Pennsylvania. To improve working conditions of migratory and other farm labor in the United States. Authorizes grants to the States for general assistance in the form of medical or any other type of remedial care.

Address communications to the A.O.A. Council on Federal Health Programs 1757 K Street, N.W. Washington, D.C.

H.R. 195 Mr. Ashley of Ohio. Amends the Social Security Act to provide insurance against the costs of hospital, nursing home, home nursing service, and diagnostic outpatient hospital services for persons eligible for OASDI benefits.

H.R. 233 Mr. Ikard of Texas. Provides income tax deduction for tuition, fees, and books to public and tax-exempt institutions of higher education.

H.R. 242 Mr. Lesinski of Michigan. Amends National Defense Education Act to provide for cancellation of student loans for service as a teacher in a private school as well as for such service in public schools.

H.R. 246 Mr. Libonati of Illinois. Establishes Bureau of Older Persons within the Department of Health, Education, and Welfare, and authorizes Federal grants to assist in the development and operation of studies and projects to help older persons.

H.R. 306 Mr. Bennett of Florida. Establishes an Office for Senior Citizens in the Department of Health, Education, and Welfare.

H.R. 357 Mr. Ford of Michigan. Amends public assistance provisions of Social Security Act to permit the payment of assistance to individuals in nonpublic mental institutions.

H.R. 424 Mr. Keogh of New York. Provides income tax deduction for expenses for transportation to and from work in the case of disabled individuals, and provides additional exemption for tax-payer or spouse who is physically or mentally incapable of caring for himself.

H.R. 447 Mr. Libonati of Illinois. Amends public assistance titles of Social Security Act so as to further assist the States in extending aid for medical care to eligible persons.

H.R. 448 Mr. Libonati of Illinois. Permits income tax deduction of all medical expenses of tax-payer or dependents, and removes limitation on deductions for medicine and drugs.

H.R. 477 Mr. McDonough of California. Permits tax-payer to carry over for three succeeding years medical expenses which exceed the maximum permissible as income tax deductions.

H.R. 531 Mr. Poff of Virginia. To provide a tax incentive for the employment of older workers.

H.R. 558 Mr. Rodino of New Jersey. Establishes a Bureau of Senior Citizens within the Department of Health, Education, and Welfare.

H.R. 616 Mr. Anfuso of New York. Provides grants-in-aid to the States for treatment of narcotic drug addicts in closed institutions, defined as hospital institutions providing a drug-free environment with maximum supervision and with minimum contact with the ouside world.

H.R. 622 Mr. Beckworth of Texas. Amends Veteran's Benefits Act to authorize the granting of hospital care to veterans in need thereof.

H.R. 628 Mr. Beckworth of Texas.

Provides that where certain veterans furnish the Administrator "statements from three doctors stating in substance that the veteran needs hospital care, the Administrator shall furnish hospital care to such veteran."

H.R. 646 Mr. Boggs of Louisiana. To protect the public health by regulating the manufacture, compounding, processing, distribution, and possession of habit-forming barbiturate and amphetamine drugs. Proposes that Congress find and declare that regulation of intrastate commerce in barbiturates and amphetamines is essential to the effective regulation of interstate commerce in such drugs, because in the form in which they are consumed their place of origin ordinarily cannot be determined.

H.R. 676 Mr. Gilbert of New York. To amend Social Security Act to provide insurance against the costs of hospital, nursing home, and surgical service for persons eligible for OASDI benefits.

H.R. 710 Mr. Lane of Massachusetts. Establishes a U. S. Office of Aging within the Department of Health, Education, and Welfare and authorizes Federal grants to assist in the development and operation of studies and projects to help older persons.

H.R. 719 Mr. Lane of Massachusetts. To provide a program of grants and scholarships to encouarge education and training in the field of nursing.

H.R. 729 Mr. McDonough of California. Amends Vocational Rehabilitation Act to provide additional Federal support to States and certain nongovernmental agencies to enable them to carry out adequate demonstration programs for the vocational rehabilitation of the physically handicapped.

H.R. 733 Mr. McDonough of California. Provides more liberal income tax treatment for amounts paid for hospitalization insurance and amounts paid under voluntary medical care plans.

H.R. 747 Mr. McDonough of California. Extends the duration of the Federal air pollution control law. Makes duration permanent.

H.R. 796 Mr. Multer of New York. Extends Federal Employees Compensation Act coverage to certain persons engaged in Civil Defense.

H.R. 805 Mr. Multer of New York. Amends the National Defense Education Act to provide for a college scholarship program.

H.R. 828 Mr. Multer of New York. To authorize the President, under certain conditions, to control, regulate, and allocate the use and distribution and price of medicinal substances, defined as any vaccine, serum, medicine, chemical, or other substances used for the prevention or treatment of disease.

H.R. 848 Mr. Teague of Texas. Places on a permanent basis the vocational rehabilitation program originally enacted as Public Law 16, 78th Congress, which provided this type of training for veterans of World War II. Passed House March 21, 1961.

H.R. 852 Mr. Teague of Texas. Authorizes the Administrator of Veterans' Affairs to establish medical advisory panels to resolve conflicts of evidence in questions involving service connection of disabilities or deaths.

H.R. 853 Mr. Teague of Texas. To insure more adequate medical care for veterans. Requires Administrator of Veterans' Affairs to maintain not less than 125,000 operating beds in hospitals to provide inpatient hospital care for an average daily patient load of not less than 113,000 patients.

H.R. 867 Mr. Teague of Texas. Provides that on and after January 1, 1966, no doctor of medicine, osteopathy, dental surgery, or dental medicine may serve as a manager of a hospital over which the Veterans Administrator has direct and exclusive jurisdiction (other than

These annual meetings of health and welfare agencies of the United States are scheduled for the coming weeks:

American Veterinary Medical Association	Detroit, August 20-24
American Hospital Association	Atlantic City, September 25-28
National Rehabilitation Association	San Francisco, October 1-3
American Academy of Pediatrics	Chicago, October 2-5
American Nursing Home Association	Cleveland, October 2-6
Arthritis and Rheumatism Foundation	New York City, October 3
American Association of Medical Record Librarians	Philadelphia, October 9-12
Muscular Dystrophy Association of America	New York City, October 10
National Association for Retarded Children	San Francisco, October 11-14

a hospital which is principally a neurological or psychiatric hospital). On and after such date no other person may serve as a manager of any such hospital unless he is a fellow of the American College of Hospital Administrators.

H.R. 870 Mr. Teague of Texas. To provide outpatient treatment for veterans of the Indian Wars on the same basis as Spanish-American War veterans.

H.R. 875 Mr. Teague of Texas. To lengthen the presumption of service connection for veterans suffering from multiple sclerosis.

H.R. 876 Mr. Teague of Texas. Lengthens the presumptive period of service connection for psychoses.

H.R. 877 Mr. Teague of Texas. Lengthens the presumption of service connection for veterans suffering from malignant tumors.

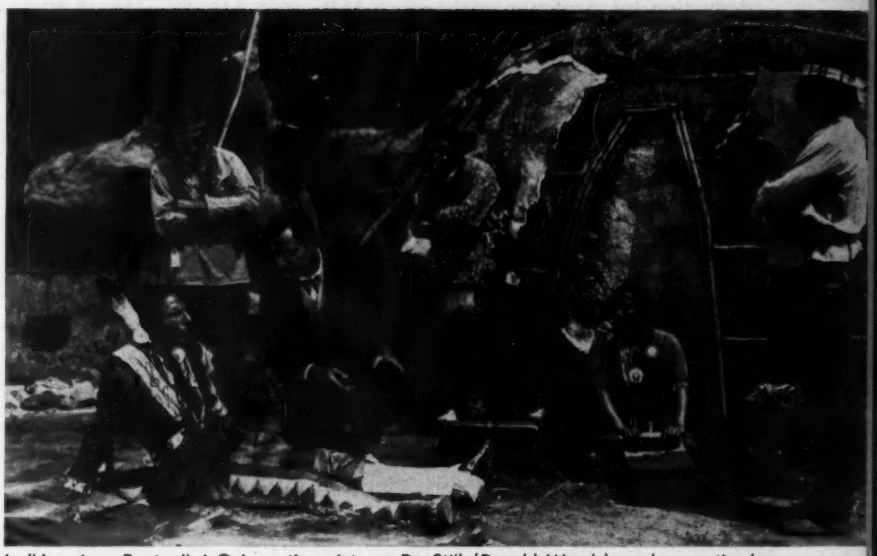
H.R. 887 Mr. Teague of Texas. Amends veterans' laws to include the reinfection type of pulmonary tuberculosis in the provision relative to presumptive service connection.

H.R. 903 Mr. Bennett of Florida. To require certain safety devices on motor vehicles sold, shipped, or used in interstate commerce.

H.R. 905 Mr. Bennett of Florida. To amend the Vocational Rehabilitation Act in order to provide assistance to the States for certain workshops, rehabilitation facilities, and rehabilitation evaluation services. Provides that in such facilities to which grants may be made all medical and related health services shall be prescribed by, or under the formal supervision of, persons licensed to practice medicine or surgery in the State.

H.R. 939 Mr. Collier of Illinois. To allow an income tax deduction for certain expenses incurred by taxpayer for the college education of a dependent.

H.R. 940 Mr. Collier of Illinois. To provide income tax credit for charitable contributions to institutions of higher education.



In "American Doctor," A.O.A. motion picture—Dr. Still (Donald Woods), as he practiced among the Indians in the early days of osteopathy.

H.R. 941 Mr. Collier of Illinois. Amends Internal Revenue Code to provide for exclusion from gross income of the proceeds of a scholarship, fellowship grant, or student assistantship without regard as to whether it represents payment for services rendered.

H.R. 951 Mr. Frelinghuysen of New Jersey. To assist institutions of higher education to market and retire bonds issued by them to finance the construction of college facilities.

H.R. 977 Mr. Huddleston of Alabama. To establish a Medical Advisory Committee on Alcoholism in the Department of Health, Education, and Welfare.

H.R. 983 Mr. Judd of Minnesota. Permits income tax deduction of amounts paid to institutions of higher education for education of taxpayer or his spouse or any of his dependents.

H.R. 1029 Mr. Monagan of Connecticut. To provide a presumption of service connection for active tuberculosis disease for peace time veterans.

H.R. 1073 Mr. Pucinski of Illinois. To provide that scholarships received by a student shall not be taken into account in determining whether he is the taxpayer's dependent for income tax purposes.

H.R. 1136 Mr. Bennett of Florida. To facilitate the procurement of doctors of medicine and doctors of dentistry for the Armed Forces by providing grants and scholarships for education in the medical and dental professions.

H.R. 1166 Mr. Healey of New York. To establish a hospital in the State of New York especially equipped for the treatment of persons addicted to the use of habit-forming drugs.

H.R. 1205 Mr. Multer of New York. To require the Surgeon General of the Public Health Service to undertake a special research program with respect to cystic fibrosis.

H.R. 1235 Mrs. Sullivan of Missouri. Amends Federal Food, Drug, and Cosmetic Act relating to labeling, special dietary foods, new drug requirements, and other provisions.

H.R. 1251 Mr. Zelenko of New York. Amends public assistance provisions of Social Security Act.

H.R. 1252 Mr. Zelenko of New York. To provide an income tax

deduction for depletion of human resources.

H.R. 1267 Mr. Zelenko of New York. To provide compensation for disability or death resulting from injury to employees in employments which expose employees to radioactive material.

H.R. 1725 Mr. Ashley of Ohio. To establish a program of scholarships for students in science and education at institutions of higher education.

H.R. 1746 Mr. Celler of New York. To authorize the establishing by the Surgeon General of an aftercare, post-hospital treatment program for drug addiction.

H.R. 1765 Mr. Dulski of New York. Amends Social Security Act for health benefits for aged. Same as H.R. 94.

H.R. 1831 Mr. Staggers of West Virginia. To authorize and request the President to mobilize in the United States an adequate number of outstanding experts to discover means of curing and preventing cancer.

H.R. 1937 Mrs. Griffiths of Michigan. To provide for the humane treatment of animals used in experiments and tests by recipients of grants from the United States and by agencies and instrumentalities of the United States Government.

H.R. 2051 Mr. Moorhead of Pennsylvania. To amend Title IV of the Housing Act of 1950 (college housing) to authorize loans to educational institutions for the construction, rehabilitation, alteration, conversion, and improvement of classroom buildings and other academic facilities.

H.R. 2083 Mr. Walter of Pennsylvania. To establish a United States Military Academy of Medicine with authority to confer the degree of Doctor of Medicine, Doctor of Dental Surgery, or Doctor of Dental Medicine upon graduates of the Academy.

H.R. 2238 Mr. Fogarty of Rhode Island. To establish a permanent bipartisan Commission to be known as the Federal Commission on Aging.

H.R. 2244 Mr. Herlong of Florida. Relating to deduction for income tax purposes of contributions to charitable organizations whose sole purpose is making distributions to other charitable organizations, contributions to which by individuals are deductible within the 30 per cent limitation of adjusted gross income. Passed the House June 14, 1961.

H.R. 2262 Mr. C. W. Miller of California. Amends Public Health Service Act to permit certain owners of fishing boats to receive medical care and hospitalization without charge at hospitals of the Public Health Service.

H.R. 2284 Mr. Rogers of Florida. To allow income tax deductions for certain payments made and expenses incurred in providing or securing higher education.

H.R. 2317 Mr. Smith of Mississippi. To provide an income tax exemption from the communications and transportation taxes for amounts paid by nonprofit hospitals.

H.R. 2318 Mr. Smith of Mississippi. To permit donations of surplus property to certain educational institutions.

H.R. 2377 Mr. Addonizio of New Jersey. Establishes Bureau of Older Persons in Department of Health, Education, and Welfare. Same as H.R. 246.

H.R. 2379 Mr. Addonizio of New Jersey. To establish the Federal Agency for Handicapped.

H.R. 2407 Mr. Dingell of Michigan. Amends Social Security Act to provide for payment of hospital and other health services furnished to aged retired individuals, and to provide for a continuing study of the health needs of such individuals.

H.R. 2414 Mr. Dingell of Michigan. To revise the Public Health Service Act, authorize grants for construction of medical, dental, osteopathic, and public health teaching facilities, provide special project grants for postgraduate public health training, and provide for Federal guaranty of loans for construction of group practice medical or dental care facilities.

H.R. 2443 Mr. Roberts of Alabama. Amends Social Security Act to provide health insurance for aged.

H.R. 2445 Mr. Roberts of Alabama. To authorize grants to assist the States in strengthening professional nurse education.

H.R. 2518 Mr. Rabaut of Michigan. Amends Social Security Act for the payment of hospital and other health services furnished to aged retired individuals.

H.R. 2705 Mr. Garland of Maine. To provide income tax deduction for certain expenses of attending colleges and universities.

H.R. 2731 Mr. Price of Illinois. Radiation Workers Compensation Act. Same as H.R. 1267.

H.R. 2747 Mr. Addonizio of New Jersey. Authorizes 10-year program of grants for construction of medical, osteopathic, dental, and public health educational facilities.

H.R. 2762 Mr. Gilbert of New York. Social Security Amendments of 1961. Same as H.R. 94.

H.R. 2764 Mr. Halpern of New York. Establishes a United States Commission on the Aging and Aged.

H.R. 2791 Mr. Price of Illinois. To provide that interest on obligations of educational institutions shall be exempt from income tax.

H.R. 2908 Mr. McDonough of California. Amends Internal Revenue Code to provide that income derived by a nonprofit hospital, medical center, or medical research organization from the lease of its facilities or equipment shall be

exempt from tax if it is used exclusively for the treatment of charity patients.

H.R. 2918 Mr. O'Konski of Wisconsin. Amends Social Security Act to provide a more liberal definition of the term "disability" for purposes of entitlement to disability insurance benefits and the disability freeze.

equipped with seat belts which meet certain safety standards.

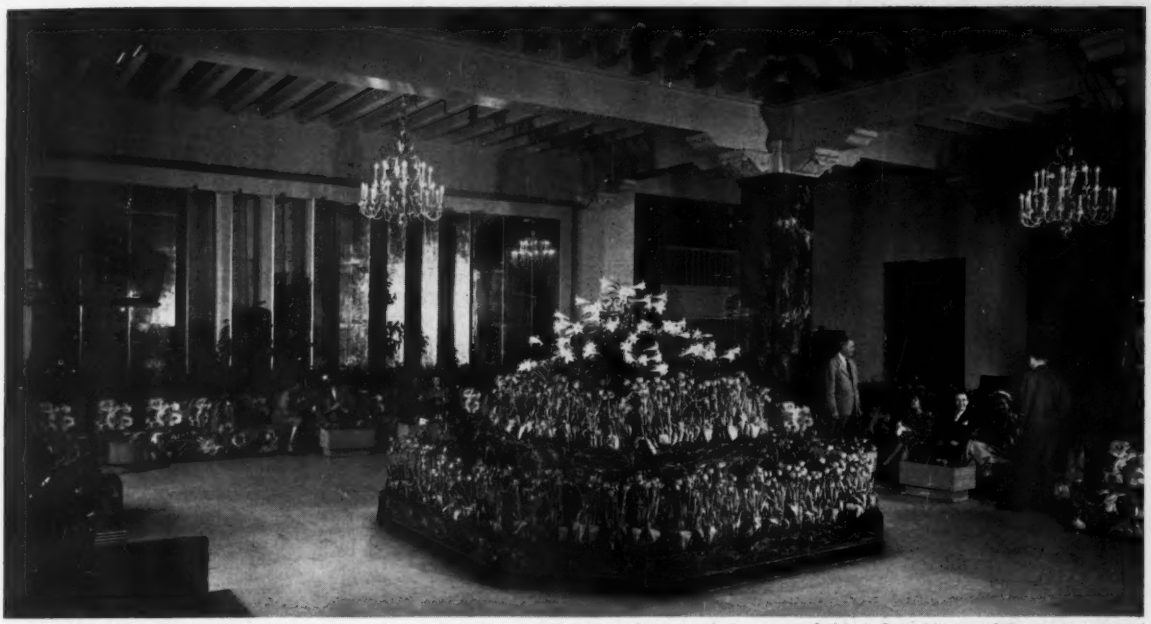
H.R. 3340 Mr. Price of Illinois. To amend the National Science Foundation Act to provide for certain educational programs.

H.R. 3438 Mr. Fogarty of Rhode Island. To provide Federal assistance to States which award scholarships to students of medicine, osteopathy, and dentistry.

Academy under the National Science Foundation.

H.R. 3587 Mr. Teague of Texas. To provide outpatient medical and dental treatment for veterans of the Indian Wars on the same basis as such treatment is furnished to veterans of the Spanish-American War. Passed House March 6, 1961.

H.R. 3739 Mr. Cramer of Florida.



Main lobby of the Drake Hotel, Chicago, meeting place for attendants at the annual meetings of the A.O.A. House of Delegates, Board of Trustees and allied groups, July 5 through 12.

H.R. 3071 Mrs. Pfost of Idaho. To establish a Senior Citizens Service within the Department of Health, Education, and Welfare.

H.R. 3162 Mr. Curtis of Missouri. To encourage basic research in science by allowance of an income tax credit for contributions and other expenditures for basic research in science.

H.R. 3276 Mr. Fogarty of Rhode Island. Amends the Public Health Service Act to provide a 10-year program of grants for education in medicine, osteopathy, and dentistry.

H.R. 3295 Mr. Kyl of Iowa. To provide that motor vehicles sold or shipped in commerce must be

H.R. 3448 Mr. Kowalski of Connecticut. Amends Social Security Act and Internal Revenue Code to provide health care insurance for the aged.

H.R. 3523 Mr. Fogarty of Rhode Island. Rehabilitation Act of 1961.

H.R. 3549 Mr. Martin of Nebraska. Provides additional income tax exemption of $1,000 for taxpayer, spouse, or dependent who is a student at an institution of higher learning.

H.R. 3573 Mr. Rodino of New Jersey. To establish a National Science

Establishes a Bureau of Senior Citizens within the Department of Health, Education, and Welfare.

H.R. 3744 Mr. Daniels of New Jersey. Rehabilitation Act of 1961.

H.R. 3967 Mr. Denton of Indiana. To protect the public health by regulating the manufacture, compounding, processing, distribution, and possession of habit-forming barbiturate and amphetamine drugs.

H.R. 3980 Mr. Harris of Arkansas. Food Additives Transitional Provisions Amendment of 1961. Public Law 87-19, approved April 7, 1961.

H.R. 4168 Mr. St. Germain of Rhode

Island. Amends Social Security Act and Internal Revenue Code to provide insurance against costs of hospital, nursing home, and surgical service for aged.

H.R. 4104 Mr. Giaimo of Connecticut. Amends Vocational Education Act relating to practical nurse training. S. 278 passed in lieu.

H.R. 4110 Mr. Halpern of New York. Authorizes a 10-year program of scholarships for medical, osteopathic, and dental education.

H.R. 4111 Mr. Halpern of New York. National Health Insurance for the Aged Act.

H.R. 4222 Mr. King of California. To provide for payment for hospital services, skilled nursing home services, and home health services furnished to aged beneficiaries under the old-age, survivors, and disability insurance program.

H.R. 4226 Mr. Bennett of Florida. Establishes a program of grants for the construction of medical, osteopathic, and dental facilities and for scholarships for medical, osteopathic, and dental students.

H.R. 4227 Mr. Boggs of Louisiana. Authorizes Secretary of Defense to initiate group medical sickness and accident insurance for retired members of the Armed Services and their dependents, which group insurance would authorize hospitalization in any civil hospital or military or Public Health Service hospital.

H.R. 4242 Mr. Flood of Pennsylvania. To provide chiropractic treatment when requested for veterans eligible for outpatient medical care.

H.R. 4247 Mr. Hebert of Louisiana. To establish an Armed Forces Medical, Dental, and Allied Sciences Academy.

H.R. 4253 Mr. Kearns of Pennsylvania. To extend for 5 years the educational programs under National Defense Education Act, and expand program.

H.R. 4259 Mr. Moorhead of Pennsylvania. Amends Housing Act relating to relocation assistance, low rent private housing for displaced families, and in connection with urban renewal areas extending benefits to hospitals in addition to colleges or universities.

H.R. 4273 Mr. Williams of Mississippi. Amends Interstate Commerce Act to provide that disabled persons may not be prohibited from operating motor vehicles in interstate or foreign commerce if such persons with certain disabilities have been examined by a doctor of medicine or osteopathy admitted to the practice of medicine or osteopathy in a State and such doctor determines that such impairment will not prevent such individuals from safely operating such vehicle.

H.R. 4343 Mr. Monagan of Connecticut. Establishes a United States Commission on the Aging and Aged.

H.R. 4351 Mr. Rodino of New Jersey. Amends National Defense Education Act to provide for scholarships for qualified college students.

H.R. 4388 Mr. Bennett of Florida. To establish a United States Armed Forces Medical Academy.

H.R. 4413 Mr. Dingell of Michigan. National Health Insurance Act.

H.R. 4571 Mr. Mills of Arkansas. To improve benefits under the old-age, survivors, and disability insurance program by increasing the minimum benefits and aged widows benefits and by making additional persons eligible for benefits under the program.

H.R. 4584 Mrs. Hansen of Washington. To provide for the same rights to non-service connected veterans of World War I as are granted to service-connected veterans of World War I in reference to medical treatment and outpatient medical services.

H.R. 4731 Mr. Curtis of Massachusetts. To provide for a program of Federal matching grants to the States to enable the States to provide health insurance for individuals aged 65 or over at subscription charges such individuals can pay.

H.R. 4777 Mr. Rivers of South Carolina. To amend the Dependents' Medical Care Act to provide that all retired members of a uniformed service qualify for benefits thereunder.

H.R. 4837 Mr. Philbin of Massachusetts. Amends Internal Revenue Code to permit certain tax-exempt organizations to engage in certain activities for the purpose of influencing legislation directly relevant to the purposes which qualify such organizations for tax exemption, without losing certain benefits under that code.

H.R. 4884 Mr. Mills of Arkansas. Amends Social Security Act to authorize Federal financial participation in aid to dependent children of unemployed parents. Public Law 87-31, approved May 8, 1961.

This section is published monthly to inform the practicing physician about new drug products and medical equipment made available on the market. It is a reference section prepared by THE JOURNAL *from descriptive material furnished by ethical manufacturers. The American Osteopathic Association does not necessarily advocate the use of these products nor disapprove any product not included. The purpose of the section is to provide trustworthy information in a convenient form.*

PRODUCT NEWS

TREPIDONE®
MEPHENOXALONE

Chemistry • Trepidone mephenoxalone is a new tranquilizer which is described as being chemically distinct from previous tranquilizing agents.

Pharmacodynamics • The drug has been reported to allay anxiety and tension without detracting significantly from mental alertness or inducing gastrointestinal discomfort. Patients taking Trepidone have shown little tendency to become detached from reality, to manifest euphoria, or to exhibit withdrawal symptoms. They generally demonstrate a normal pattern of response to everyday situations without undue excitability or apprehension.

Toxicology • Side effects occur infrequently at normal dosage levels and are the same as those associated with other non-phenothiazine tranquilizers: dizziness, drowsiness, nausea, and rashes. Clinical and experimental investigations have failed to reveal toxic effects even at higher than recommended dosage.

Indications • Indications for Trepidone include a wide range of anxiety and tension states of low to medium intensity. It is also indicated as an adjunct to other measures in the treatment of physical disease complicated by psychogenic factors. It is useful for the reduction of muscle spasm.

Dosage schedule • The recommended dosage for adults is one tablet four times a day; children's doses are proportionately smaller. Higher than tranquilizing dosages may be required for the treatment of muscle spasm.

How supplied • Tablets of 400 mg., in bottles of 50.

Manufacturer • Lederle Laboratories, Division of American Cyanamid Company, Pearl River, New York.

NACTISOL T.M.

Chemistry • Nactisol is a combination of Nacton poldine methylsulfate (chemically designated as 2-benziloyloxymethyl-1,1-dimethylpyrrolidinium methylsulfate) and Butisol Sodium (butabarbital sodium) chemically designated as sodium 5-sec-butyl-5-ethylbarbiturate).

Pharmacodynamics • Nactisol is specifically designed to inhibit excess gastric acid secretion and decrease gastrointestinal spasm in the nervous or anxious patient. The Nacton poldine methylsulfate component is thought to inhibit gastric acid secretion at the source, which is the parietal cell; thus the total output of hydrochloric acid is reduced by about 50 per cent for at least 6 hours following administration of the drug. Nacton also reduces gastrointestinal motility. Butisol Sodium provides smooth, predictable sedation for 5 to 6 hours in the nervous patient.

Absorption; fate; excretion • The rate and manner of metabolism of the drug permit the body to handle therapeutic doses with little likelihood of cumulative effects, building of tolerance, or toxic reactions. In laboratory animals, Butisol Sodium was found to be metabolized at about 6 per cent per hour; it is almost entirely destroyed in the body, with very little excreted unchanged.

Toxicology • In chronic toxicity studies in animals, Nacton given orally every day for 6 months brought no demonstrable changes in behavior, general appearance, or food consumption. There were no significant effects on blood, kidney, or liver, no histologic changes, and no deaths. Side effects noted in the clinical use of Nactisol have been minimal. Slight dryness of the mouth, dizziness, heartburn, and sleepiness have occasionally been reported.

Indications • Nactisol is especially useful in reducing the hypersecretion associated with peptic ulcer and hyperacidity. Clinically, Nactisol significantly reduces gastrointestinal motility and may be of benefit to patients with ulcerative colitis, gastritis, hyperchlorhydria, enteritis, dumping syndrome, duodenitis and pancreatitis. Nactisol is most useful when tension, anxiety, or an emotional overlay complicates treatment of disturbances of gastric acid secretion and gastrointestinal motility.

Contraindications • As with any agent containing a potent anticholinergic, careful consideration should be given before administering Nactisol to patients with glaucoma or potential glaucoma, prostatic hypertrophy, pyloric obstruction, coronary artery dis-

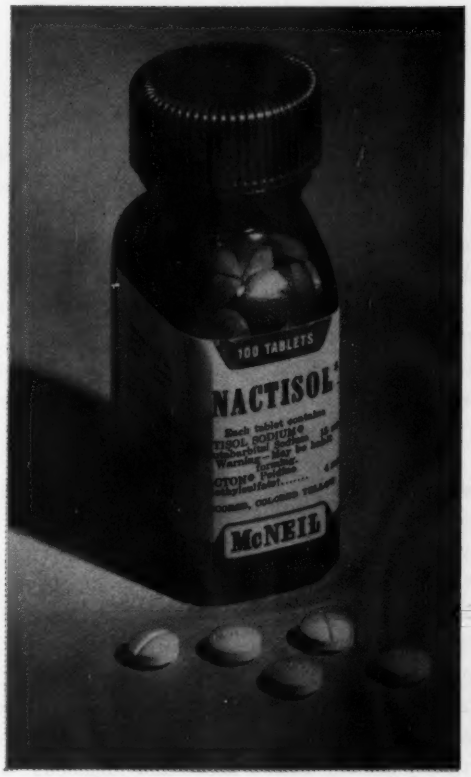
ease, or tachycardia. When Nactisol therapy is interrupted it should be resumed at a reduced dosage and gradually increased to the patient's therapeutic level.

Dosage schedule • The average effective dose of Nactisol is 1 tablet three or four times daily, before meals and at bedtime. However, the dose should be individualized for best therapeutic results.

How supplied • Nactisol is supplied in scored, yellow tablets, imprinted "McNeil," in bottles of 100. Each tablet contains 4 mg. of Nacton poldine methylsulfate and 15 mg. of butabarbital sodium.

Manufacturer • McNeil Laboratories, Inc., 2900 North 17th Street, Philadelphia 32, Pennsylvania.

References • Steigmann, F., Lorber, S. H., and Rider, J. A., Clinical Reports to McNeil Laboratories.



DEXALONE DURA-TABS AND DEXATAL DURA-TABS

Chemistry • Dexalone is dextro-amphetamine sulfate, a sympathomimetic amine; Dexatal combines dextro-amphetamine with phenobarbital. The Dura-Tab formula provides sustained-release medication.

Pharmacodynamics • These dietary adjuncts help obese patients to lose weight, by controlling appetite and relieving the emotional distress which often induces overeating. Dexatal, in addition to controlling appetite and improving mood, provides gentle sedation to help allay restlessness, tension, or insomnia which may accompany dieting. The effects of dextro-amphetamine sulfate are mediated through selective action on the central nervous system.

Toxicology • Dextro-amphetamine seldom gives rise to side effects. When Dexatal is used, the habit-forming potential of phenobarbital should be taken into account.

Indications • Dexalone Dura-Tabs are indicated for obese patients who simply need help in curbing their appetites; Dexatal Dura-Tabs are specifically helpful in cases complicated by emotional factors.

Contraindications • Dextro-amphetamine sulfate is contraindicated in the presence of hyperexcitability and agitated prepsychotic states. It should be used with caution in the presence of cardiovascular or coronary disease, severe hypertension, or hypersensitivity to sympathomimetic amines.

Dosage schedule • The usual dose of either medication is one Dura-Tab before breakfast.

How supplied • Dexalone *10* and Dexalone *15* Dura-Tabs contain 10 mg. and 15 mg. respectively of dextro-amphetamine sulfate; Dexatal *10* and Dexatal *15* Dura-Tabs contain, in addition to 10 and 15 mg. of dextro-amphetamine, 30 mg. and 45 mg. respectively of phenobarbital.

Manufacturer • Wynn Pharmacal Corporation, Philadelphia 31, Pennsylvania.

SURBEX-T T.M.

Chemistry • Surbex-T Filmtabs provide therapeutic dosage of the B-complex vitamins plus vitamin C for oral administration when water-soluble vitamins are depleted or demands are increased.

Pharmacodynamics • Surbex-T supplies the water-soluble vitamins lacking in cases of moderately severe deficiencies or in which pathologic conditions have either reduced the biologic availability or increased the need for these vitamins.

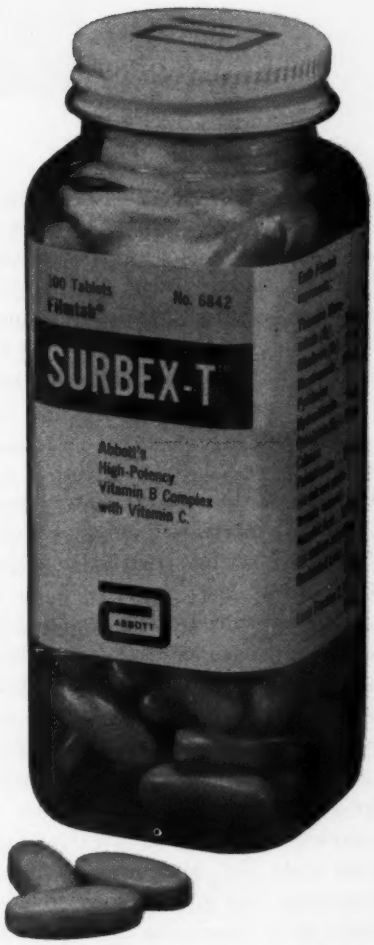
Indications • Surbex-T is indicated for acute or chronic illnesses such as cardiovascular conditions, gastrointestinal disorders, liver disorders, hyperthyroidism, and cachetic states; before or after surgery; for severe burns, fractures, or infections; during prolonged oral administration of antibiotics; during radiation therapy; for restrictive diets following depletion caused by illness; and for depletion due to alcoholism.

Contraindications • No contraindications to this vitamin preparation have been reported.

Dosage schedule • The usual dose is 1 Filmtab daily, this dose to be varied according to individual needs.

How supplied • Surbex-T Filmtabs are supplied in bottles of 100 and 1,000.

Manufacturer • Abbott Laboratories, North Chicago, Illinois.



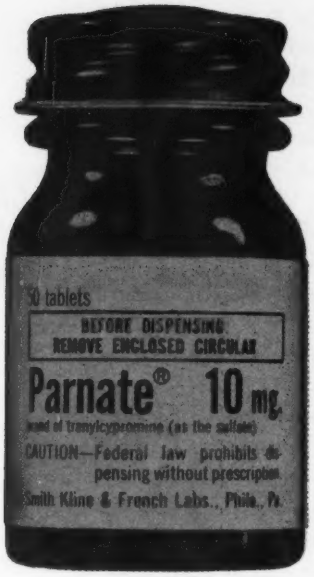
PARNATE®

Chemistry • Each Parnate tablet contains 10 mg. of tranylcypromine (trans-*dl*-2-phenylcyclopropylamine) as the sulfate.

Pharmacodynamics • Parnate is thought to influence higher subcortical areas of the central nervous system, possibly the hypothalamus. It is a potent and unusually rapid inhibitor of monoamine oxidase (MAO), an enzyme system which regulates the levels of serotonin, norepinephrine, and other neurohumoral amines in brain tissue. The increase in these amines resulting from MAO inhibition is believed to facilitate transmission of impulses within the brain and thus to relieve mental depression indirectly. The antidepressant effect of Parnate differs in kind from that achieved by direct stimulation of the central nervous system; depressed patients display not only greater energy but renewed interest, restored ability to concentrate, and relief of morbid thought patterns. The onset of action with Parnate has been reported to be more rapid than with other similar antidepressants.

Absorption; fate; excretion • Animal studies show that Parnate is readily absorbed and rapidly metabolized, although the organs involved in this rapid detoxification have not yet been identified. In rats given radioactive Parnate orally, more than 70 per cent of the drug was excreted in the urine within 24 hours; small amounts were excreted in the feces, but none was expired as carbon dioxide. Moreover, because Parnate is not a hydrazine derivative, the accumulation of free hydrazine during metabolic breakdown (a possible cause of jaundice) cannot occur.

Toxicology • Long-term toxicity studies in animals have revealed no tissue damage attributable to the drug. Side effects of Parnate are usually mild; incidence is closely related to dosage. The effects that have been reported with standard dosages include restlessness, overstimulation, insomnia, weakness, drowsiness, dizziness, dry mouth, nausea, diarrhea, abdominal pain, constipation, and occasionally headache. Symptoms of postural hypotension have been noted, usually in patients with pre-existent hypertension. Other side effects which might occur in rare instances are tachycardia, urinary retention, significant anorexia, skin rash, edema, palpitations, blurred vision, tinnitus, chills, paresthesia, muscle spasm and tremors, impotence, and sweating. Transitory hypertension might occur in rare cases. Most of these side effects can usually be relieved by adjusting the dosage or giving suitable concomitant medication.



Indications • Parnate is indicated for the relief of such symptoms as dejected mood, self-depreciation, lowered activity, difficulty in making decisions, disturbed eating and sleeping patterns, and variations of these symptoms. Efficacy has been demonstrated in reactive and other psychoneurotic depressions as well as in psychotic depressive disorders.

Contraindications • Since Parnate is specific only for depressive symptoms, it may aggravate anxiety or increase agitation in cases with mixed symptoms. In such cases the dosage should be reduced or a phenothiazine tranquilizer administered concurrently. There are no absolute contraindications to the use of Parnate, but the following precautions are important: Patients inclined to suicide should be treated with Parnate only when custodial measures are available; electroconvulsive therapy is often preferable in such cases. The drug should not be used for patients with a history of liver disease or with abnormal liver function, pending further clinical experience. In patients with impaired renal function, the possibility of cumulative effects of the drug should be borne in mind. Combinations of Parnate and other potent drugs such as phenothiazine derivatives should be used with caution. In particular, Parnate and imipramine should not be given together or in immediate succession. Caution should be observed in giving Parnate to patients with recent myocardial infarction or coronary artery disease with angina of effort, and also patients with epilepsy.

Dosage schedule • The recommended starting dose for office patients is 20 mg. per day, divided in two doses, morning and afternoon. If no response is noted after 2 to 3 weeks, the dosage is increased to 30 mg. daily by adding 10 mg. to the morning dose. Doses higher than this should generally be used only if the patient is hospitalized or under comparable supervision. After satisfactory response, the dosage may be reduced to a maintenance level—often 10 mg. daily.

How supplied • Parnate is available in coated, rose-colored tablets, each containing 10 mg. of tranylcypromine as the sulfate, in bottles of 50.

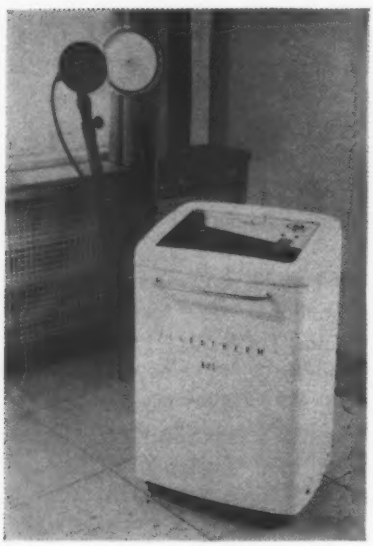
Manufacturer • Smith Kline & French Laboratories, Philadelphia 1, Pennsylvania.

References • Lemere, F., Am. J. Psychiat. 117:249, 1960. Agin, H. V., Am. J. Psychiat. 117:150, 1960. Scherbel, A. L., A.M.A. Arch. Int. Med. 107:37, 1961. Petersen, M. C., Scientific exhibit, A.P.A., Atlantic City, 1960. Roebuck, B. E., and Maccubbin, H. P., Scientific exhibit, A.M.A. Clinical Meeting, Dallas, 1959.

SHORT-WAVE APPARATUS

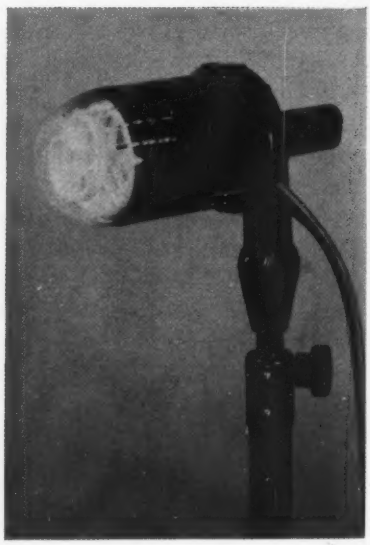
Description • The Ultratherm apparatus for short-wave therapy, developed in Germany by the Siemens-Reiniger-Werke Ag., incorporates the following new features in diathermy equipment: deep-heating efficiency, automatic tuning, and microwave-equivalent therapy by means of the specially designed Monode and Minode electrodes. The special circuit designed for deep-heating efficiency allows for the fact that short-wave therapy necessitates considerable distance between electrode and skin, and that the apparatus should be able to deliver full output to the patient over such distances. Of equal importance is the proper tuning of the short-wave apparatus. Conventional stabilizers and other devices have not solved this problem, since they must be constantly adjusted by hand during treatment, and any slight movement of the patient may disturb the tuning and therefore the output. The Siemens automatic tuner, however, tunes in the machine and keeps it tuned during treatment, guaranteeing precise and reliable output. To achieve the simple application and high thermic effects of microwave therapy, two coil-field electrodes have been specially designed, Monode for large areas and Minode for special applications in cavities, et cetera. These electrodes enable the practitioner to adapt the short-wave therapy apparatus for microwave-equivalent therapy. Microwave therapy does not replace short-wave diathermy, especially for deep-heating, but with either Monode or Minode, microwave therapy can be achieved where necessary. All other electrodes, of course, can be used with the Ultratherm apparatus.

Manufacturer • The Siemens Physical Medicine Division is represented in the United States by the Physical Medicine Division, Midwest Imports, P.O. Box 322, Hinsdale, Illinois.



ULTRATHERM 603

MONODE



MINODE

CURRENT LITERATURE

Parenteral fluid regimens in the treatment of severe diarrhea in infants

► A comparative study of three methods of treatment for infant diarrhea is reported by Harris L. Smith, M.D., and James N. Etteldorf, M.D., in *The Journal of Pediatrics,* January 1961. The study was based on 53 cases of severe diarrhea in children less than 15 months old. Before and during treatment, serial observations were made of the clinical effects, blood pH, and plasma levels of base bicarbonate, sodium, and potassium. Comparison of results showed that the modified Hartmann method was most effective in promptness of relief of severe acidemia and acidosis, but frequently produced alkalemia and alkalosis during the second day of treatment. Deficits of potassium were not adequately corrected; in a high percentage of the patients these deficits were aggravated. Levels of plasma sodium frequently rose above the normal range in patients whose pretreatment values were normal or subnormal. In infants treated by the Butler-Talbot plan, acidemia, acidosis, and hyponatremia persisted for long periods. Low levels of plasma potassium rapidly rose to normal, and subnormal pretreatment levels of potassium often were elevated above normal range. The moderately elevated levels were not accompanied by clinical evidence of hyperkalemia. In infants treated by the Darrow regimen, levels of blood pH and plasma base bicarbonate were slower in returning to normal than were those in patients treated by the modified Hartmann method. Acidemia and acidosis were not so prolonged, however, as in the group treated by the Butler-Talbot regimen. Tendencies to overshoot normal electrolyte levels were not evident, and low levels of plasma sodium and potassium were effectively corrected. Hypernatremic infants in all three groups responded similarly with respect to clinical course and electrolyte levels; in all of these patients, the levels were slow in returning to normal.

It appears that the gaps in our understanding of the physiology of body fluids in health and disease still preclude the formulation of an ideal regimen of parenteral fluid therapy for severe diarrhea in infants. The authors feel that comparative studies of current methods will help the practitioner in evaluating their relative efficacy and in modifying them for use in specific situations.

Puerperal hematomas

► An analysis of 112 cases of puerperal hematoma and a review of pertinent literature are included in this article by Paul Pedowitz, M.D., Samuel Pozner, M.D., and Nathan H. Adler, M.D., in the *American Journal of Obstetrics and Gynecology,* February 1961. Suspecting that this complication occurs more often than has been realized, the authors set out to establish a realistic incidence for the disease, to review and summarize the clinical aspects, to evaluate current treatment methods, and to emphasize that this complication is inherent in normal labor. The 112 cases in this series, observed at the Jewish Hospital of Brooklyn from January 1933 to June 1958, represent about one sixth of all the cases described in medical literature, of which more than half have been reported since 1954. It seems clear, therefore, that the majority of cases have never been reported. Although numerous factors predispose to the formation of hematoma, the trauma inherent in normal labor is the only factor common to all cases and is the specific etiologic agent. Thus, hematomas are generally unavoidable and are not a reflection on the obstetrician's capabilities. The authors suggest two measures leading to earlier recognition of puerperal hematoma: (1) routine inspection of vulva and perineum immediately postpartum, to detect hematoma in the vulvar and episiotomy sites, and (2) immediate vaginal examination of all patients complaining of severe perineal pain during the early postpartum period. This will usually disclose the paravaginal and vulvovaginal hematomas before there is extensive disruption of the perineum and loss of blood. Delay in diagnosis is responsible for many of the more extensive perineal disruptions. Immediate surgical repair is the treatment of choice when feasible; in other cases,

evacuation of the hematoma and packing of the cavity, followed by secondary repair, are indicated. Blood replacement and adequate antibiotic prophylaxis are essential. In cases of subperitoneal or supravaginal hematoma, laparotomy is sometimes necessary, with hysterectomy or ligation of the hypogastric artery in order to secure hemostasis. Under modern conditions, death from puerperal hematoma is very rare; also, infection of the hematoma almost never occurs. Although the incidence of this complication has been reported in a range from 1:561 to 1:4,000 deliveries, the authors estimate the true incidence to be about 1:300 to 1:500.

Roentgen signs of pneumoperitoneum in the newborn

▶ The roentgen signs of pneumoperitoneum in the newborn are reviewed and illustrated in an article by Enrique Schwarz, M.D., in *The American Journal of Roentgenology, Radium Therapy, and Nuclear Medicine*, April 1961. Delayed diagnosis in such cases frequently leads to the infant's death. Perforation can occur anywhere along the gastrointestinal tract, frequently producing pneumoperitoneum. A careful study of the roentgenograms in such cases discloses the presence of free air in the abdomen, thus establishing the diagnosis and reducing the risk of death. Emphasis is placed on the less commonly recognized findings seen on roentgenograms taken in the supine and lateral positions. The earliest roentgenographic sign of pneumoperitoneum is that of air underneath the diaphragm, which can be detected only when the films are made with the patient in the erect position, and the diagnosis may be missed when films are taken with the infant on his back. Pneumoperitoneum should be suspected, however, if the supine view shows the presence of a central abdominal translucency; without such awareness this sign may be mistaken for intestinal dilation. The sign can be confirmed by erect roentgenograms; the same results can be obtained if the infant is inverted and held by its feet, when free air will tend to fill the pelvic cavity. On lateral roentgenograms, the semilunar collection of air anterior to the abdominal viscera is considered characteristic of free air in the peritoneal cavity. Familiarity with such signs assists the radiologist in establishing an early diagnosis.

An adjustable lower-extremity brace

▶ An adjustable brace to facilitate investigation and management of gait problems in certain muscle disorders is described by Thomas L. DeLorme, M.D., Laird Johnson, B.S., Peter Luchini, B.S., William Bora, M.D., and James Morrissey, M.D., in *The Journal of Bone and Joint Surgery*, March 1961. The device consists of calf and thigh bands, two adjustable side-bars or uprights, joints at the knee and ankle, and a shoe clamp to attach the brace to the heel of the shoe. Each component is independently adjustable, making the appliance adaptable to either the right or left leg and to all types of patients except those who are unusually tall, short, or fat. A special brace is used for children. When assembled as a long brace, the apparatus weighs 6 pounds; when used as a short brace it weighs 3 pounds. In more than a year of use, only one mechanical failure was encountered; the duraluminum tongue of the knee lock broke and was replaced by a stainless-steel tongue, with which there has been no trouble.

A quickly adjustable brace has obvious advantages in many situations. In the early stages of some neurologic diseases the muscle function may change rapidly, necessitating frequent adjustment in the supportive apparatus. In long-standing disorders with abnormal gait patterns, the best device for a given patient can often be determined only after a series of trials and errors. During convalescence from reconstructive procedures, short-term use of a brace is often desirable but not justified because of the high cost of a conventional brace. Also, when a permanent brace is being fabricated, temporary use of the adjustable brace allows rehabilitation to continue without interruption. Moreover, a temporary brace can substantially reduce the number and extent of alterations required before a permanent brace is finally acceptable to the patient.

Although there are some disadvantages with this brace, its adaptability can be increased by minor changes in design. The importance of this particular device is that it demonstrates that an adjustable brace is technically feasible, practical, and of clinical value.

Postinjection sciatic nerve palsies in infants and children

▶ A suggestion that the gluteal injection site be abandoned in small or debilitated patients is embodied in a review by Floyd H. Gilles, Lt. (MC) USNR, and Joseph H. French, M.D., in *The Journal of Pediatrics*, February 1961. Sciatic palsies secondary to injection into the gluteal mass have been recognized for at least 40 years. The pathogenic mechanisms that have been suggested include allergic peripheral neuritis, puncture of the nerve, ischemia of the nerve, and direct neural damage by drugs. In the 21 cases reviewed in this article, there was no clinical evidence of hypersensitivity to the medication used; penicillin and tetracycline were the only agents directly implicated, although multiple drugs were involved by association. In the authors' opinion, direct injection of neurotoxic material into or near the nerve is the most probable mechanism of sciatic nerve damage. In infants or debilitated older children it is extremely easy to inject into or near the sciatic nerve.

Divergent views have been set forth regarding the treatment of postinjection sciatic palsies in in-

fants and children. It now appears that surgical intervention, involving neurolysis, should not be considered unless there is a deep granuloma in the affected buttock or unless prolonged conservative care has failed to bring satisfactory improvement. The authors' experience indicates that in the absence of a deep granuloma, neurolysis should not be performed until after 7 to 12 months of treatment with no improvement.

More important than treatment, however, is the question of prevention. Since inadvertent angulation of the needle in an uncooperative infant may result in injection of potentially neurotoxic agents into or near the sciatic nerve, it is suggested that a safer injection site be chosen. It is pointed out that the lateral aspect of the thigh, at the juncture of the distal third and proximal two thirds, offers a relatively large muscle mass without any important underlying neurovascular structures, and this area is therefore recommended as a preferable injection site.

Spontaneous pneumothorax with a review of ninety-five cases

► A study undertaken to obtain a more objective evaluation of spontaneous pneumothorax and its management is reported by Richard H. Adler, M.D., Samir Marrash, M.D., and F. Niguidula, M.D., in the *New York State Journal of Medicine*, February 15, 1961. The discussion is directed to the practicing physician, for he is the one who must make the correct diagnosis and institute proper treatment.

In the 95 cases reviewed, 78 of the patients were male. Ages ranged from 1 day to 80 years, but the incidence was highest in the 20- to 40-year age group. Also noted was a progressive increase in the number of hospital admissions per year. In recent years there has been increasing recognition of spontaneous pneumothorax, and advances in thoracic surgery have stimulated a change from the policy of conservative observation to a more aggressive approach in an effort to obtain rapid and permanent lung re-expansion. In the authors' experience, conservative treatment may be indicated at the time of a patient's first episode, provided there is less than about 25 per cent lung collapse. Close observation is necessary to make certain that the air leak is not progressive and that complications such as tension pneumothorax and bleeding do not develop. When the collapse is greater than 25 per cent, prompt evacuation of the air is indicated; the authors recommend insertion of an intercostal tube, after using the needle to confirm the location of the free intrapleural air space. The tube is more effective than repeated needle aspirations, and when the free air is removed quickly the lung usually re-expands within hours or a few days. It is important to realize that recurrences are more frequent than have been thought, and will occur in 5 to 45 per cent of the patients. The authors feel that after two documented episodes on one side, the third occurrence should be treated by open thoracotomy with excision of the offending lesion and parietal pleural stripping. Also, if blebs or bullae can be demonstrated either by x-ray or thoracoscopy, thoracotomy is indicated.

The diagnosis in infants rests mainly on cyanosis; in older patients, pain is the most common symptom. Chest x-ray films always should be taken to confirm the clinical impression and establish the diagnosis. The classical case of spontaneous pneumothorax in an otherwise healthy young male is almost always due to rupture of a subpleural bleb or vesicle, not uncommonly associated with areas of scarring as in old healed tuberculosis. The onset is not necessarily related to strain or exertion.

Reconstruction for loss of an eyebrow

► A quick and simple method for reconstructing an eyebrow is described by John B. Erich, M.D., in the *Proceedings of the Staff Meetings of The Mayo Clinic*, March 15, 1961. Although most patients are not overly sensitive about the cosmetic defect resulting from loss of an eyebrow, the deformity is conspicuous and its correction is worth while. In the author's experience, the use of a full-thickness graft from the scalp offers a practical means of reconstructing an eyebrow. The whole procedure can be completed within 2 weeks. If the bed of tissue in which the graft is to be inlaid is excessively scarred, it may be preferable to use a pedicle flap of hair-bearing scalp tissue for the reconstructive procedure; however, this method requires months, and the cosmetic effect is not likely to be satisfactory. The free full-thickness graft should be tried in practically all patients, and the pedicle-flap method resorted to if the graft fails to heal.

Treatment of fractures and dislocations of the cervical spine, complicated by cervical-cord and root injuries

► A comparative study of fusion vs. nonfusion therapy in fractures of the cervical spine is reported by Donald Munro, M.D., in *The New England Journal of Medicine*, March 23, 1961. Evidence is presented that in cases of cervical-spine fracture-dislocations, with or without cord or root complications, treatment by internal fixation and operative fusion of the bones involves higher mortality, more undesirable symptoms, and less effective results than treatment by skeletal traction without either internal fixation or fusion. For the return of stability to the cervical spine after injury, provision must be made for the repair or torn ligaments. This provision must include release of associated muscular spasm, reduction of dislocations to a practical anatomic position, removal of compression of the cord, and immobilization until the ligaments are healed. Skeletal traction, carefully applied, is the

treatment of choice. Plaster of Paris is contraindicated, in the care of cervical fracture-dislocations complicated by a cord injury. Early operation for bony fusion is also contraindicated, but delayed operation is permissible and probably mandatory when it becomes clear that paralysis of the paraspinal muscles has developed. Delayed operative bony fusion, if indicated, is optional for the correction of post-therapeutic root pain; otherwise the procedure has little to offer. Internal fixation by wiring is not helpful and may actually have an injurious effect on the mechanics of the cervical spine.

The author re-emphasizes the validity of the concept, first stated in 1939, that spinal cord injuries are different from spinal column injuries without cord involvement, and that all significant cord injuries are accompanied by spinal shock and its effects. Surgical treatment of spine injuries carries a measurable risk of death of the patient, or of producing injury to the cord in cases where such injury was not present before operation. The author's experience, together with an analysis of data from 359 cases, leads to the conclusion that major surgical procedures are almost never necessary for future stability and rehabilitation of the injured spine.

Reconstruction of the common bile duct by end-to-end anastomosis without the use of an internal splint or stent support

► An experimental and clinical study of surgical procedures affecting the common bile duct is reported by John L. Madden, M.D., and William J. McCann, M.D., in *Surgery, Gynecology & Obstetrics*, March 1961. The study was prompted by doubts about the necessity for the temporary or prolonged use of a foreign body substance, such as a T tube, as an internal splint or stent support following reconstructive operations. In reviewing the history of such operations, it was observed that the first successful results were obtained with end-to-end anastomoses without internal splint or stent support. After about 1913 the T tube came into increasing use and eventually was considered mandatory in connection with an end-to-end duct reconstruction, either immediate or delayed. Moreover, it has generally been believed that the tube must remain in place for at least 6 to 9 months, for best results. The authors feel, however, that the use of the T tube is empiric and not justified by the available evidence, which shows that results without an implanted tube have been just as good as or perhaps better than those obtained with the T tube.

In this study 73 operations were performed on 56 dogs separated into 4 experimental groups according to type of operative procedure. The clinical study included 8 patients in whom reconstruction of the common duct was carried out by end-to-end anastomosis without an internal splint or stent support. Later it was learned that a similar series, involving 7 patients, had been treated and studied in the same way at another institution. Results in the two series were combined in this article, so that the report covers 15 patients, among whom there were 13 successful operations, 1 failure, and 1 unexplained death which occurred suddenly on the day after the operation. After reviewing the experimental and clinical data, the authors conclude that primary reconstruction of the common bile duct by end-to-end anastomosis, without an internal splint or stent support and without tension on the suture line, is a logical and feasible operation.

Treatment of acute alcoholism with methocarbamol

► Skeletal muscle hyperactivity in acutely alcoholic patients can be relieved by the intravenous administration of methocarbamol, according to a report by Joseph J. Lofaro, M.D., in the *New York State Journal of Medicine*, March 15, 1961. When alcoholic patients are hospitalized, the physician in charge must consider their requirements from the standpoint of medical and nursing care and length of stay in the hospital. Intravenous fluids and vitamins quickly overcome dehydration and vitamin deficiencies, and sedatives induce adequate sleep. Under this regimen alone, however, the patient continues to be incapacitated by agitation, tremor, and tension. To test the effect of skeletal muscle relaxtion on the progress of patients undergoing treatment for acute alcoholism, a study of 100 patients was carried out with the relaxing agent methocarbamol (Robaxin), because of its assured efficacy and freedom from serious side effects. The intravenous preparation was considered ideal for this purpose because many patients in this situation are unable to tolerate oral medications easily. Also, the drug in this form can be added to other intravenous fluids often administered to severely dehydrated alcoholic patients.

The regimen consisted of 1 gram of methocarbamol intravenously on the first day, 2 grams the second, and 3 on the third day. During the next 2 days the patients received oral methocarbamol, 2 grams four times a day, and on the sixth and seventh days the total oral dosage was reduced to 6 grams. Muscle hyperactivity and tremor were completely controlled in 95 patients, and only slight tremor persisted in three others after 7 days of treatment. The effects in two patients could not be evaluated because of acute delirium tremens. No serious side effects occurred at these dosage levels; mild reactions such as drowsiness, dizziness, and warmth were transient. The effective reduction in skeletal muscle hyperactivity enabled the patients to return to a satisfactory level of self-care in less time than was possible under the usual regimen of symptomatic treatment.

BOOK REVIEWS

► *Books for review which were received during the period from May 5 to June 5 are listed on pages A-138 to A-150. Reviews of these books will be published as space permits.*

► AN INTRODUCTION TO OSTEOPATHIC MEDICINE. By W. V. Cole, M.A., D.O., F.A.C.N. Cloth. Pp. 276, with illustrations. Price $5.50. Kansas City College of Osteopathy and Surgery, 2105 Independence Avenue, Kansas City 24, Missouri, 1961.

An addition to the slim shelf of books about osteopathy is always welcome when its object is the advancement of the profession. Particularly needed are those books which seek to explain the bases of osteopathic theory and practice.

It has been said that the only way to understand osteopathy is to share the experience of osteopathic physicians. While this may be true in an absolute sense it poses serious handicaps to beginning students who must seek to learn osteopathy and its broad medical context at the same time. Thus, the new edition of Dr. Wilbur V. Cole's syllabus for entering students should be valuable to them and to anyone else seeking a basic understanding of osteopathy.

By necessity, Dr. Cole comes immediately to the need for defining and explaining much about osteopathy which remains unproved and controversial. All his assertions may not—indeed could not—please all osteopathic scholars, but they do provide a foundation upon which shared experience can be built.

If he does not succeed in defining precisely the "osteopathic concept," he does succeed in explaining how it concerns the over-all health of the body. In many instances (for example, "vertebral articular strain" for "osteopathic lesion"), his terminology is an effort to improve on traditional usage.

The book's contents are arranged in classic and usable form. The author begins with a general historical review of medicine and introduces osteopathy in the context of the nineteenth century American frontier. He attempts no apologies for the "service" nature of early osteopathic instruction and the relative neglect of research into the nature of Dr. Still's theory and technics.

Though he admits that the whole thesis remains to be proved scientifically, he musters clinical and logical evidence to support the fundamental assertion that the vertebral articular strain exercises an abnormal reflex activity which may affect visceral function.

Dr. Cole is emphatic in basing his explanations on sound anatomy. He points out that the principal virtue of manipulative treatment lies in its ability to "normalize" body function by interrupting troublesome reflex patterns. This is a controllable factor in the ecologic approach to the patient which is the real fundamental of osteopathic practice.

On page 37, he lists three current principles of osteopathy which have been evolved from the original and somewhat contradictory writings of Dr. Still. These, he says, are a basis for modern concepts of osteopathy.

1. Every portion of the body must have an adequate arterial supply and venous drainage,
2. Within each individual there are normally present the chemical substances necessary to maintain the individual in a state of health, and
3. When the structure of a cell, tissue or organ is altered, the function is also altered.

In considering these statements, it should be stressed that the duration and extent of the alterations (or disease process) are important when attempting to evaluate the ability of the organism to recover. When the natural inherent recovery potential is lost, the organism cannot recover.

In some detail, Dr. Cole elaborates on the nature of the vertebral articular strain, showing the way in which primary, secondary, and compensatory strains have an effect on total health. The utilization of manipulative therapy, as he explains, is a part of the total approach to health and disease which other medical teaching is only beginning to emphasize.

The author has drawn from osteopathic sources and general medical references to provide extensive footnotes and bibliography for each of his nine chapters. The presentation reflects many years of study and assimilation of materials.

In summary, Dr. Cole accomplishes his stated objectives of reviewing the place of osteopathy in the historical march of medicine, describing the vertebral articular strain and its effect on health

and disease, and detailing the work done to support and utilize the osteopathic concept. His book, as a text, will supply a recognized need in a creditable manner.

▶ BLOOD DISEASES OF INFANCY AND CHILDHOOD. By Carl H. Smith, M.A., M.D., Professor of Clinical Pediatrics, Cornell University Medical College, New York, N.Y.; Attending Pediatrician, The New York Hospital, New York, N.Y.; Consulting Pediatrician, Beekman-Downtown Hospital, New York, N.Y.; Fitkin Memorial Hospital, Neptune, N.J.; Misericordia Hospital, New York, N.Y.; New York Infirmary, New York, N.Y.; St. Joseph's Hospital, Far Rockaway, N.Y.; Sea View Hospital, Staten Island, N.Y.; Consulting Hematologist in Pediatrics, Lennox Hill Hospital, New York, N.Y. Cloth. Pp. 572, with illustrations. Price $17.00. The C. V. Mosby Company, 3207 Washington Boulevard, St. Louis 3, 1960.

This book represents a major attempt at organizing into a single volume the expanding literature pertaining to pediatric hematology. The format is orthodox, with discussions of the origin and development of blood cells, the normal values and changes occurring in the blood during infancy and childhood, and then a more detailed discussion of the individual hematologic disorders relative to the leukocytes, erythrocytes, reticuloendothelial system, and the coagulation mechanism. For the most part the book is clinically oriented, therapy being well detailed and integrated with laboratory and clinical features of the disease or syndrome. Sections are devoted to the pathogenesis of the major entities under discussion, and the author's wide personal experience is frequently drawn upon to illustrate points.

An occasional statement may be disputed; however, these statements usually represent the author's opinion and, when read and interpreted in that light, are not to be belabored. It would seem the indicated purpose of the book has been realized for the most part. The readability of the presented materials, its organization, and bibliographic documentation are good. The use of both author and subject indexes is always valuable. The binding and printing are of high quality, enhancing the value and durability of the book. All practitioners dealing with infants and children will do well to have this as a reference text.

SIDNEY J. KATZ, D.O.

▶ NERVE ENDINGS IN NORMAL AND PATHOLOGIC SKIN. Contributions to the Anatomy of Sensation. By R. K. Winkelmann, M.D., Ph.D., Section of Dermatology, Mayo Clinic, Rochester, Minnesota. Cloth. Pp. 195, with illustrations. Price $7.50. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Illinois, 1960.

I am glad of the opportunity to bring this arresting little book to the attention of osteopathic readers, for it deserves a wider immediate reading public than it will likely get. Addressed to a limited audience—it is one of the Thomas series of *American Lectures in Dermatology*—it presents a stimulating challenge to physiologists, anatomists, biologists, neurologists, pathologists, and medical thinkers almost everywhere. It is of deep interest to me, an osteopathic general practitioner, because it annihilates some of the comfortable concepts of anatomy and neurophysiology I have acquired over the years.

Backing his statements with ample documentation, notably that of neurophysiologist G. Weddell, and by some intensive research of his own, Winkelmann offers good evidence that, among other eponymic items, neither von Frey's hot and cold spots, nor the classic organs of Ruffini (heat), or end-bulbs of Krause (cold), exist. As for Müller's law of specific nerve energies, at least as far as the skin goes, it may be considered repealed. And regarding Herbst's corpuscles—well, they are indeed genuine, but in avian anatomy only.

But the author does more than shatter one's eponymic dreams; he reduces the anatomy of the sensory apparatus of the skin to what would appear to be its least common denominator: that of an all-embracing neural meshwork with certain peripheral extensions, plus the pacinian corpuscles. With these two basic entities, the author goes on to discuss how the modalities to touch, pain, heat, and cold can theoretically be accounted for by the one, and the well-documented faculty of pressure sensitivity by the other.

This monograph is easy to read, and short—about 160 pages of text, including scores of mostly original photographic illustrations. He begins in the preface with "an effort to record a complete pattern of nerve endings of the skin of man," and closes, after quoting Weddell, that "absolutely no morphologic basis is present for the theory of von Frey concerning the reception of a specific sensation by a specific structure." Then he states flatly, "It is time that this hypothesis is discarded completely." In between these statements the author figuratively fills his pen with methylene blue, and spells out with vital stain the doom of some favorite histologic myths.

It may be argued: The doctrine of specific nerve energies has been a conveniently workable one; is this new hypothesis of a nonspecific multipurpose sensory nerve net workable, and can it be proved? To which, with Weddell, the author might reply: With the possible exception of the pressure-sensitive pacinian corpuscles there are no specific nerve-ends. There are only adaptations in degree of multipurpose nerves to constitutional needs and to local environmental stresses within the skin. (All, for instance, respond in some degree to the stimulus of touch.) Here are the morphologic facts; "prove" what you want with them.

Winkelmann accounts for the rich variety of sensory nerve-end apparatuses that have been described, as results of unsatisfactory methods of preparation, especially in the early days; examination of an insufficient number of serial sections; lack of standardization of technics; and inadequate equipment. It occurs to me that perhaps, too, the early investigators may have been some of the post-

Renaissance inheritors of the Aristotelian passion for minutely classifying things. "So many observers," Winkelmann quotes H. H. Wollard, "so many endings."

Winkelmann, acknowledging Weddell, offers the spatiotemporal concept of sensory interpretation in the skin. This growing concept is based on a process of analysis and synthesis, the mechanism of which is concerned with differentials in spatial stimuli and in conduction frequencies.

The book contains a chapter on nerves in cutaneous disease, which the author admits is inadequate, because of lack of systematic investigation by pathologists and dermatologists. There is also a chapter and an appendix listing the author's favorite methods of staining tissues, including the intra-arterial vital methylene blue method, a technic which has been utilized in much of the recent research on the skin.

H. G. GRAINGER, D.O.

► HANDBOOK OF PHYSIOLOGY. By R. J. S. McDowall, M.D., D.Sc., M.R.C.P. (Lond.), F.R.C.P. (Edin.), Emeritus Professor of Physiology, University of London. Ed. 43. Cloth. Pp. 759, with illustrations. Price $12.50. Hazell Watson and Viney Ltd., Aylesbury and Slough, England. J. B. Lippincott Company, exclusive U.S. distributors, East Washington Square, Philadelphia 5, 1960.

This is a textbook that has grown up with physiology, since its first edition in 1848. The constant effort of the present author has resulted in nine of the book's forty-two editions being published in the past 23 years.

As the changing means of research have brought to light new physiologic explanations of processes that were not understood in detail, or their interpretations were modified, this British author has filtered out the pertinent facts for medical students. In this edition, the author has revised the section on gastric secretion, cardiac output, the pituitary, and muscle contraction. The section on the pituitary body is of particular interest to physicians who use Acthar and other steroids.

This book is recommended to students, particularly those who have had adequate preliminary courses in physiology, and to physicians who, over the years, have only subconsciously been thinking in physiologic terms. Marked changes have taken place, and this text will bring physicians up to date.

A. P. ULBRICH, D.O.

► PSYCHOTHERAPISTS IN ACTION. Explorations of the Therapist's Contribution to the Treatment Process. By Hans H. Strupp, Department of Psychiatry, School of Medicine, University of North Carolina, Chapel Hill. Cloth. Pp. 338, with illustrations. Price $8.75. Grune & Stratton, Inc., 381 Fourth Avenue, New York 16, 1960.

In this book the author is concerned with the behavior of therapists rather than with the therapeutic process or a situation. The one always involves the other, but it takes much more courage to do a study of a therapist than of a situation. The author is aware of the grave difficulty which besets an investigator when dealing with the therapist-variable. This is the task he has chosen, a task fraught with value judgments, an admission he is at pains to make in his preface: "I am fully aware that value judgments dictated parts of the statistical analysis (for example the explorations of the therapist's empathy)."

A strange logic pervades the work of many competent researchers in the field of psychotherapy. They seem to think that if an idea, a process, a situation, or a person does not lend itself to valid and reliable scientific treatment, it has to be relegated, with due apologies, to the area of the "subjective." By implication, nonscientific material is suspect, and is to be either discarded or merely tolerated like the poor but ever-present relative. The error in this strange logic—it is strange because investigators who use it are always exemplary in their logical precision—is to suppose that we cannot be intelligent or reasonable about nonscientific experience. I would expect that concepts not readily conducive to scientific control would require all the more careful description and practical definition. If definition is reserved for precise scientific treatment, then at least we may expect a clear working description of the value-loaded material in question, if it appears that such material will be used in the proposed study.

Thus the central weakness in this serious and conscientious study is the use of a value-loaded concept, central to the investigation, which is not clearly described or differentiated from many other vaguely similar terms. This concept is empathy. It appears as a key concept in the outline of his research—Part 1—and much of what later is recorded in his statistical analysis depends upon it. "It emerged that differences in the degree of empathy shown by the therapist-respondents appear to be a considerably more fruitful indication of their total approach and structural differences to which the system gives major emphasis" (page 243). The author, in a footnote (page 303), is aware that empathy "has numerous and fuzzy meanings," and refers the reader to the operational criteria used to judge the presence or absence of empathy in Chapter IV.

Anyone experienced in therapy may well agree that the author's "criteria" (pages 78 and 79) are fruitful observations of therapists who get along well with patients. But the question arises: Are these criteria of empathy, or of sympathy, or of rapport, or of common, human decency between two people in communication? The reviewer believes that the skills of empathy are intricate and different from community of feeling, from sympathy, from rapport, and from mutual interpersonal decencies, though all these may well be involved in an act of empathy.

The dynamics of identification, the emergence of imitation, the play of choice, the clarifying act of identity both of therapist and patient—all these need to be described if we are to arrive at a concept

of empathy that can be distinguished from community of feeling, from sympathy, or from rapport. Is the empathic act deliberate or spontaneous? Does it have elements of both design and impulse? Of the various sources of the concept of empathy, what are the important leads? Is the therapist to be conscious of clear aims in the use of empathy, or is he empathic by chance or talent?

There is as much confusion in the use of the term "empathy" as there is in meanings of "identification." Neither identification nor empathy lends itself easily to scientific treatment. Each tends to carry with it value implications, but it is no criticism of empathy and identification to note their complexity, their nonscientific components, and their value overtones. Indeed, it is all the more necessary to be as clear as possible about them, when they reveal that they embrace more than scientifically manageable processes. Even more elusive, but just as much in need of careful, extensive description are the persons who reveal empathic ability—empathic persons as distinct from sympathetic, permissive, or understanding persons.

The author has attempted a worth-while task—the need to study the persons who give therapy, as well as technics, situations, and patients. Much of his observation is timely in a period when psychology is rather stifled by its rigid methodology. His courage might be stimulated to go one step further. That would be to treat his nonscientific material by a method better suited to it rather than to cram his value-loaded concept into the Procrustean bed of scientific categories and measurement. The author uses, to illustrate his point, client-centered psychologists and three groups of psychiatrists: group I, of no experience; group II, of intermediate experience; and group III, of extensive experience. He then discusses the ways of communication used in each instance, that is, the amount of activity, the amount of exploration, the amount of clarification, discussion, interpretation, direct guidance, and other miscellaneous channels.

In many ways this is an excellent study. It is unique and, for those engaged in any practice of psychotherapy, it is very informative.

THOMAS J. MEYERS, PH.D., D.O.

► THE TRANSPLANTATION OF TISSUES AND ORGANS. By Michael F. A. Woodruff, M.D., M.S. (Melb.), F.R.C.S., F.R.C.S.E., F.R.A.C.S., Professor of Surgical Science in the University of Edinburgh; Surgeon-in-Charge, The Royal Infirmary, Edinburgh; formerly Ralph Barnett Professor of Surgery in the University of Otago, Dunedin, New Zealand. Cloth. Pp. 777, with illustrations. Price $25.50. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Illinois, 1960.

This rather monumental work is probably the only current compendium of information on the subject of tissue and organ transplantation. However, much has been written by many scattered workers; evidence enough of this can be found in the fact that the author has collected over 4,000 references, all of which he has listed in this book.

The book itself has discussions on basic principles and technics, autotransplants, typical behavior of homotransplants, factors which may determine long survival of homotransplants, homagraft immunity, the effect of various experimental procedures on the behavior of homotransplants, specific immunologic tolerance of homotransplants, heterotransplants, storage and sterilization of tissues, transplantation as an instrument of research (genetics, experimental embryology, physiology, and experimental pathology), transplantation of tissues and organs in surgery, skin grafting (free grafts and flaps), transplantation of adipose tissue, connective tissue and tendon, voluntary muscle, nerve, bone, cartilage, cornea, blood vessels, hemopoietic tissue, endocrine tissues, kidney, lung, heart, spleen, and liver, and pedicle transplantation and anastomosis of hollow viscera, including the alimentary tract and the urinary tract.

► P-Q-R-S-T. A Guide to Electrocardiogram Interpretation. By Joseph E. F. Riseman, M.D., Assistant Clinical Professor of Medicine, Harvard Medical School; Visiting Physician, Beth Israel Hospital, Boston, Massachusetts. Ed. 4. Cloth. Pp. 168, with illustrations. Price $6.50. The Macmillan Company, 60 Fifth Avenue, New York 11, 1960.

Four difficulties are encountered by the beginner in electrocardiography: (1) lack of knowledge; (2) lack of a logical, step by step, method for interpretation of each tracing; (3) lack of recognition of the value and especially of the limitations of electrocardiography; and (4) lack of experience. This book, now in its fourth edition, is designed to offer help in the first three areas. It is not intended to replace standard texts, but rather to serve as a "bedside manual"—to provide clues to diagnosis so that reference to standard texts will be more meaningful.

An interesting feature is the clear statement of the limitations and values of this diagnostic method. Little or no information is provided by the electrocardiogram about valvular damage, the functional capacity of the heart, or the prognosis in the individual case. This seemingly magical instrument cannot predict the future or replace clinical knowledge, experience, and judgment. It cannot point out where the wide variations in normality become abnormalities, nor can it be held responsible for its own technical inaccuracies. On the other hand, the electrocardiogram is the final authority in the diagnosis of most cardiac arrhythmias, and it yields important information about the state of the ventricular myocardium—thus furnishing invaluable guidance in diagnosis and management of certain cases.

The author has made liberal use of charts and diagrams to make this complex diagnostic method more understandable. His verbal descriptions also have diagrammatic clarity, a boon to any learner.

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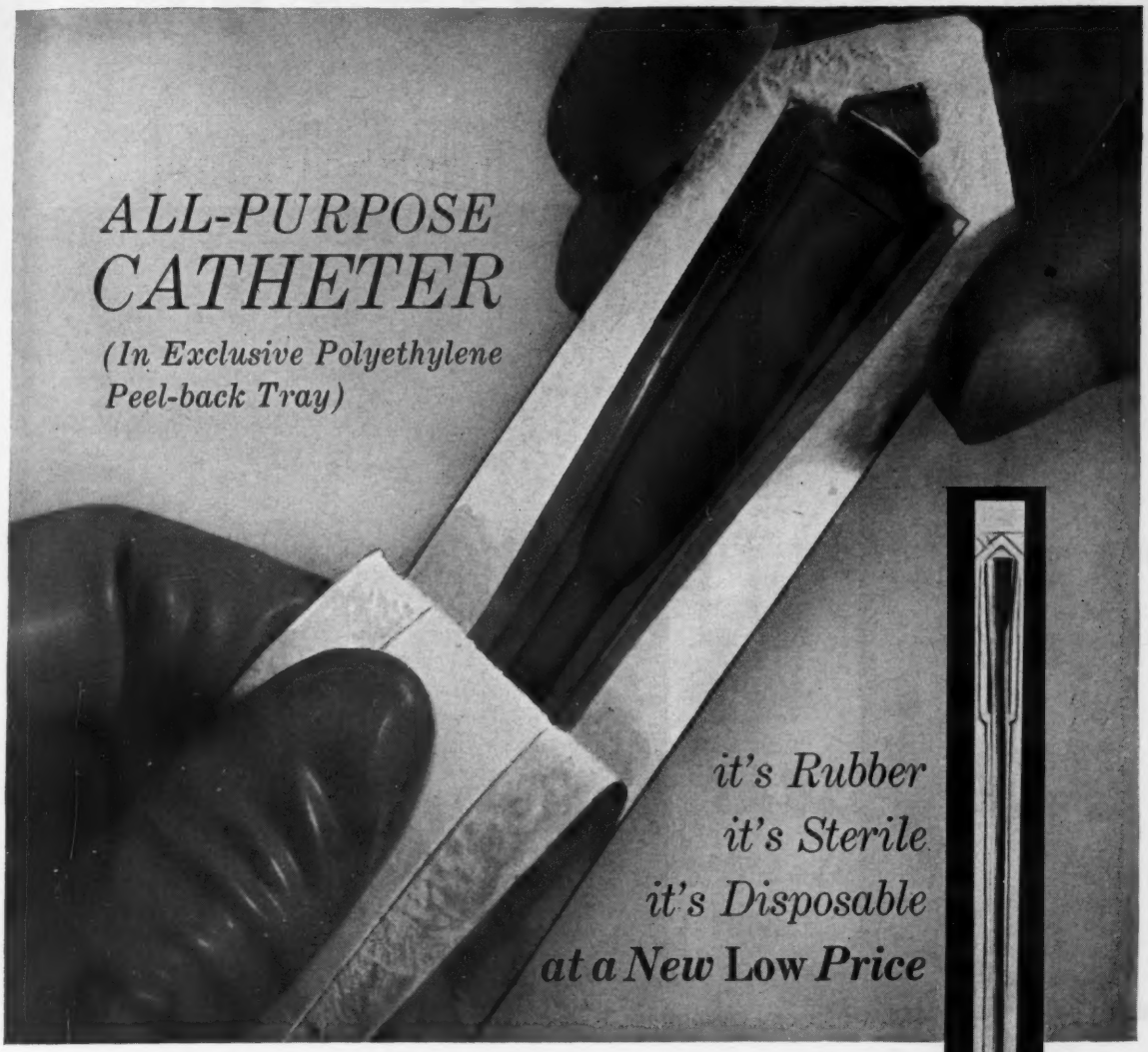

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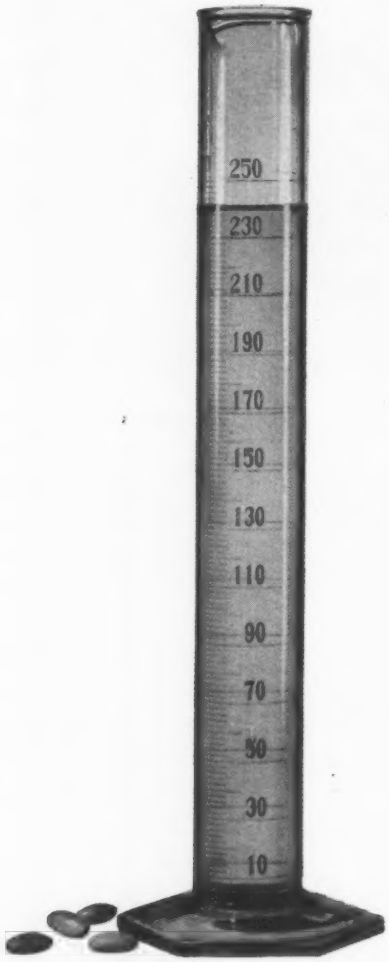

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American College of Osteopathic Obstetricians and Gynecologists and the American College of Osteopathic Pediatricians, annual meeting, Americana Hotel, Bal Harbour, Fla., February 19-22. A.C.O.O.G. Program Chairman, John C. Dickinson, Jr., 3272 West Rd., Trenton, Mich. Secretary, Arthur A. Speir, Box 66, Merrill, Mich. A.C.O.P. Program Chairman, Arthur Snyder, 386 N. E. 167th St., North Miami Beach, Fla. Secretary, Martyn E. Richardson, 9553 Lackland Rd., St. Louis 14.

American College of Osteopathic Surgeons, annual clinical assembly, Denver Hilton Hotel, Denver, Colo., October 29-November 2. Executive Secretary, Charles L. Ballinger, Box 40, Coral Gables, Fla.

American Osteopathic Association, Sixty-Sixth Annual Convention, Las Vegas, Nev., January 15-18. Program Chairman, W. Clemens Andreen, Andreen Clinic, 1475 Ford Ave., Wyandotte, Mich.

Canadian Osteopathic Association, annual meeting, Lord Simcoe Hotel, Toronto, October 5-7. Program Chairman, Norman W. Routledge, 15 Ursuline Ave., Chatham, Ont. Secretary, Miss Joyce S. Currie, 609 Medical Arts Bldg., Montreal 25, Quebec.

Central States Osteopathic Society of Proctology, annual meeting, Oliver Hotel, South Bend, Ind., September 22-24. Program Chairman, William J. Blacker, 48 W. 28th St., Grand Rapids 9, Mich. Secretary, Lloyd B. Hoxie, 39011 Harper, Mt. Clemens, Mich.

District of Columbia, see **Maryland.**

Eastern States Osteopathic Society of Proctology, annual meeting, Marriott Motor Hotel, Washington, D.C., November 4-5. Program Chairman, Felix D. Swope, 1028 Connecticut Ave., N.W., Washington 6, D.C. Secretary, LeRoy W. Lovelidge, Jr., 201 E. Orange St., Lancaster, Pa.

Florida, annual meeting, Carillon Hotel, Miami Beach, October 2-4. Executive Director, Mr. Barton K. Johns, 5009 Central Ave., Tampa 3.

Hawaii, annual meeting in combination with Western States Osteopathic Society of Proctology, Princess Kaiulani Hotel, Honolulu, October 9-12. Program Chairman, Alan R. Becker, 1739 Kalakaua Ave., Honolulu 14. Secretary, Joseph M. Thomas, 1541 S. Beretania St., Honolulu 14.

Idaho, see **Washington.**

Kansas, annual meeting, Broadview Hotel, Wichita, September 23-27. Program Chairman, Irwin J. Conant, Box 125, Meriden. Executive Secretary, Mr. Lloyd L. Hall 823 W. 10th St., Topeka.

Louisiana, annual meeting, Hotel Monteleone, New Orleans, October 12-14. Program Chairman, Carl E. Warden, 827 Hodges St., Lake Charles. Secretary, V. L. Wharton, 406-07 Weber Bldg., Lake Charles.

Maryland, District of Columbia, and Virginia, annual meeting, Carvel Hall, Annapolis, Md., October 7-8. Program Chairman, E. R. MacDonald, 1318 West St., Annapolis. Secretary, Joseph W. Lindstrom, 2732 St. Paul St., Baltimore 18.

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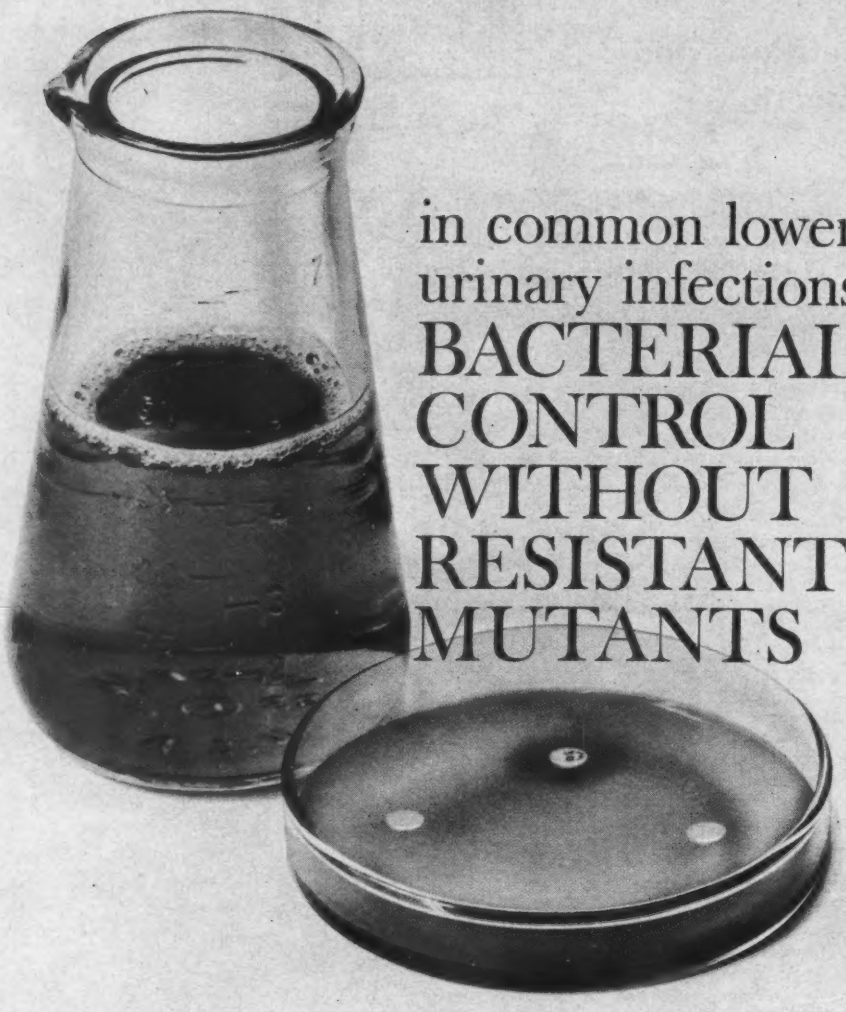

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Missouri, annual meeting, Sheraton-Jefferson Hotel, St. Louis, October 3-5. Program Chairman, Harry S. Still, Still-Hildreth Osteopathic Sanatorium, Macon. Executive secretary, Mr. Paul D. Adams, 325 E. McCarty St., Jefferson City.

New Jersey, clinical conference, Cherry Hill Inn, Delaware Township, September 24. Executive Secretary, Mr. R. P. Chapman, 1212 Stuyvesant Ave., Trenton 8.

New Mexico, annual meeting, Western Skies Hotel, Albuquerque, April 12-15. Program Chairman, George C. Widney, Jr., 1125 Kent Ave., N. W., Albuquerque. Executive Secretary, Mr. L. Thomas Christison, P.O. Box 696, Albuquerque.

New York, annual meeting, Sheraton Atlantic Hotel, New York City, October 13-14. Program Chairman, George F. Johnson, 353 77th St., Brooklyn 9. Executive Secretary, C. Fred Peckham, 38 E. Bridge St., Oswego.

North Carolina, annual meeting and refresher course, Battery Park Hotel, Asheville, October 26-28. Program Chairman, Joseph H. Huff, Box 1177, Burlington. Secretary, Walter C. Eldrett, 310 S. Main St., Hendersonville.

Oregon, see **Washington.**

Osteopathic College of Ophthalmology, and Otorhinolaryngology, annual meeting, Americana Hotel, Bal Harbour, Fla., September 26-28. Executive Secretary, Arthur A. Martin, Box 222, Kirksville, Mo.

Pennsylvania, midyear refresher course, Hotel Hershey, Hershey, October 13-15. **Annual meeting,** Penn Sheraton Hotel, Pittsburgh, May 17-19. Executive Secretary, Mr. Thomas M. Fogarty, 1941 Market St., Harrisburg.

Vermont, annual meeting, Mt. Snow, Dover, September 27-28. Program Chairman, Mason B. Barney, Box 336, Manchester Center. Clerk, Marian N. Rice, 8 Court St., Windsor.

Virginia, see **Maryland.**

Western States Osteopathic Society of Proctology, annual meeting, in combination with Hawaii Association of Osteopathic Physicians and Surgeons, Princess Kaiulani Hotel, Honolulu, October 9-12. Program Chairman, George W. Diver, Box 180, Elsa, Texas. Secretary, Marcus S. Gerlach, 2015 State St., Santa Barbara, Calif.

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Washington, Oregon, and Northern Idaho, annual meeting, Olympic Hotel, Seattle, Wash., June 7-9. Secretary, Lon A. Hoover, 1219 Sixth Ave., Tacoma 5.

Ontario, annual meeting, Park Hotel, Niagara Falls, May 7-9. Program Chairman, A. Reid Johnston, 1298 King St., E., Hamilton. Secretary William K. Church, 85 Neywash St., Orillia.

State and national boards

Alaska basic science examinations given on request. Address R. Harrison Leer, M.D., secretary, Board of Examiners, Alaska Office Bldg., Juneau.

Arizona Those interested in professional examinations should contact Russell Peterson, D.O., secretary, Osteopathic Board of Registration and Examination in Medicine and Surgery, 2747 E. McDowell Rd., Phoenix 22.

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Basic science examinations September 19 at the University of Arizona, Tucson. Address Millard G. Seeley, Ph.D., secretary, Board of Examiners in the Basic Sciences, University of Arizona, Tucson.

Colorado basic science examinations in September. Address Esther B. Starks, D.O., secretary, Basic Science Board, 1459 Ogden St., Denver 18.

Connecticut basic science examinations October 14. Address Miss M. G. Reynolds, executive assistant, Board of Healing Arts, 110 Whitney Ave., New Haven 10.

District of Columbia examinations in October. Address Mr. Paul Foley, deputy director, Department of Occupations and Professions, 1740 Massachusetts Ave., N.W., Washington 6, D.C.

Hawaii For information on examination dates write to Richard Y. Kodama, D.O., secretary, Board of Osteopathic Examiners, 826 Keeaumoku St., Honolulu 14.

Illinois examinations in October. Applications must be filed by the middle of September. Address Mr. Frederic B. Selcke, superintendent, Department of Registration and Education, Capitol Bldg., Springfield.

Iowa basic science examinations October 10 at the Capitol Building, Des Moines. Address Elmer W. Hertel, Ph.D., secretary, Board of Basic Science Examiners, Wartburg College, Waverly.

Maryland examinations in October. Address Christopher L. Ginn, D.O., secretary, Board of Osteopathic Examiners, 19 W. Mulberry St., Baltimore 1.

Michigan examinations September 12-14. Applications must be filed by September 1. Address Roy G. Bubeck, Jr., D.O., secretary, Board of Osteopathic Registration and Examination, 2851 Clyde Park Ave., S.W., Grand Rapids 9.

Basic science examinations in October. Address Mrs. Anne Baker, secretary, Board of Examiners in the Basic Sciences, 116 Mason Bldg., Lansing.

Minnesota basic science examinations October 3 at the University of Minnesota, Minneapolis. Address Raymond N. Bieter, M.D., secretary, Board of Examiners in the Basic Sciences, 105 Millard Hall, University of Minnesota, Minneapolis 14.

Montana examinations September 5. Address Warren E. Monger, D.O., secretary, Board of Osteopathic Examiners, Box 64, Dillon.

Nebraska basic science examinations October 3-4. Address Mr. R. K. Kirkman, director, Bureau of Examining Boards, Department of Health, State Capitol Bldg., Lincoln 9.

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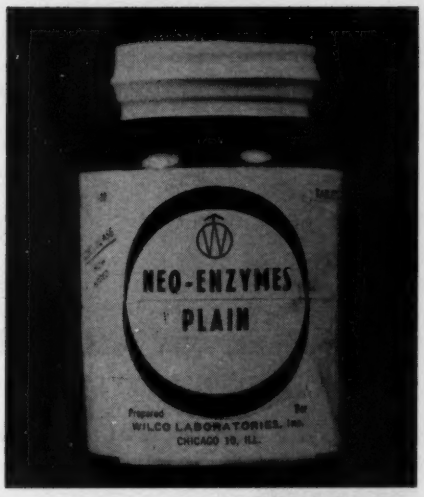

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New Hampshire examinations September 13-16 at Concord. Address Edward W. Colby, M.D., secretary, Board of Registration in Medicine, State House, Concord.

New Jersey examinations October 17-20. Applications must be filed 20 days in advance. Address Vincent Butler, M.D., secretary, Board of Medical Examiners, 28 W. State St., Trenton 8.

New Mexico basic science examinations July 16. Address Mrs. Marguerite Cantrell, secretary, Board of Examiners in the Basic Sciences, Box 1522, Santa Fe.

North Dakota examinations in July. Address M. J. Hydeman, D.O., secretary, Board of Osteopathic Examiners, 417½ Broadway, Bismarck.

Oregon examinations in July. Address Mr. Howard I. Bobbitt, executive secretary, Board of Medical Examiners, 609 Failing Bldg., Portland 4.

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* Hall, E. W., "The Family Doctor & Preventive Dentistry" G.P. XXII:6, Dec. 1960

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ber 5. Address Mr. Joaquin Mercado Cruz, secretary, Board of Medical Examiners, c/o Department of State, Box 3271, San Juan.

Rhode Island examinations October 5-6. Address Mr. Thomas B. Casey, Administrator of Professional Regulations, 366 State Office Bldg., Providence.

Basic science examinations August 9. Address Mr. Casey.

South Dakota examinations July 18-19. Address Mr. John C. Foster, executive secretary, Board of Medical and Osteopathic Examiners, Room 300, First National Bank Bldg., Sioux Falls.

Tennessee examinations in July at Nashville. Address M. E. Coy, D.O., secretary, State Board of Examination and Registration for Osteopathic Physicians, 1226 Highland, Jackson.

Basic science examinations given every 3 months. Address O. W. Hyman, M.D., secretary, Board of Basic Science Examiners, 62 S. Dunlap, Memphis 3.

Utah examinations third week in September. Applications must be filed 10 days prior to examinations. Address Mr. Frank E. Lees, director, Registration Division, Department of Business Regulations, State Capitol, Salt Lake City 14.

Washington examinations in July. Address Mr. Thomas A. Carter, secretary, Professional Division, Department of Licenses, Olympia.

West Virginia examinations in October. Address Donald C. Newell, D.O., secretary, Board of Osteopathy, 137½ Main St., Oak Hill.

Wyoming examinations October 2 at Cheyenne. Address James W. Sampson, M.D., secretary, Board of Medical Examiners, State Office Bldg., Cheyenne.

Alberta examinations in September at Edmonton. Address Mr. G. B. Taylor, acting registrar, Office of Registration, University of Alberta, Edmonton.

Saskatchewan examinations in October at Saskatoon. Address Anna Northup-Little, D.O., 2228 Albert St., Regina.

Reregistration of osteopathic licenses

Within 60 days after July 1—Indiana, $5.00 residents; $10.00 nonresidents. Address H. D. Wolf. D.O., treasurer, Board of Medical Registration and Examination, 4840 N. Michigan Rd., Indianapolis.

September 1—Nebraska, $3.00. Address Mr. R. K. Kirkman, Director of Bureau of Examining Boards, State Department of Health, Lincoln 9.

September 1—Ohio $2.00. Address H. M. Platter, M.D., secretary, State Medical Board, 21 W. Broad St., Columbus 15.

Examination by National Board

The National Board of Examiners for Osteopathic Physicians and Surgeons, Inc. conducts Parts I and II of its examinations on the first Thursday and Friday of each May and December at the six approved colleges. Application blanks may be obtained from the secretary of the Board or the dean of the college, and the completed application blank, together with check for the part to be taken, must be in the secretary's office by the November 1 or April 1 preceding the examination.

Examinations in Part I consist of anatomy, including histology and embryology; physiology; physiological chemistry; general pathology; and bacteriology, including parasitology and immunology.

Part II consists of examinations in surgery, including applied anatomy, surgical pathology, and surgical specialties, neurology and psychiatry; public health, including hygiene; medical jurisprudence; obstetrics and gynecology; pediatrics; os-

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Part III is an oral and practical examination given under the supervision of a chief examiner who is a member of the Board and by a panel of associate examiners. Subjects covered in Part III are: anatomy; physiology; pathology; osteopathic principles, therapeutics and pharmacology; surgery; ophthalmology and otorhinolaryngology; obstetrics and gynecology; physical and clinical diagnosis; public health and communicable diseases.

These are oral examinations which the candidate may take after having satisfactorily completed the first six months of a 1-year internship in a hospital approved for intern training. Part III is given annually.

The next examinations in Part III will be given in the spring of 1962. Dates will be announced in the November issue of THE JOURNAL.

All candidates are reminded that the examinations must be completed within a period of 7 years. Candidates who took Part I in 1955 must take Part III in 1962 or forefeit the right to complete the examinations.

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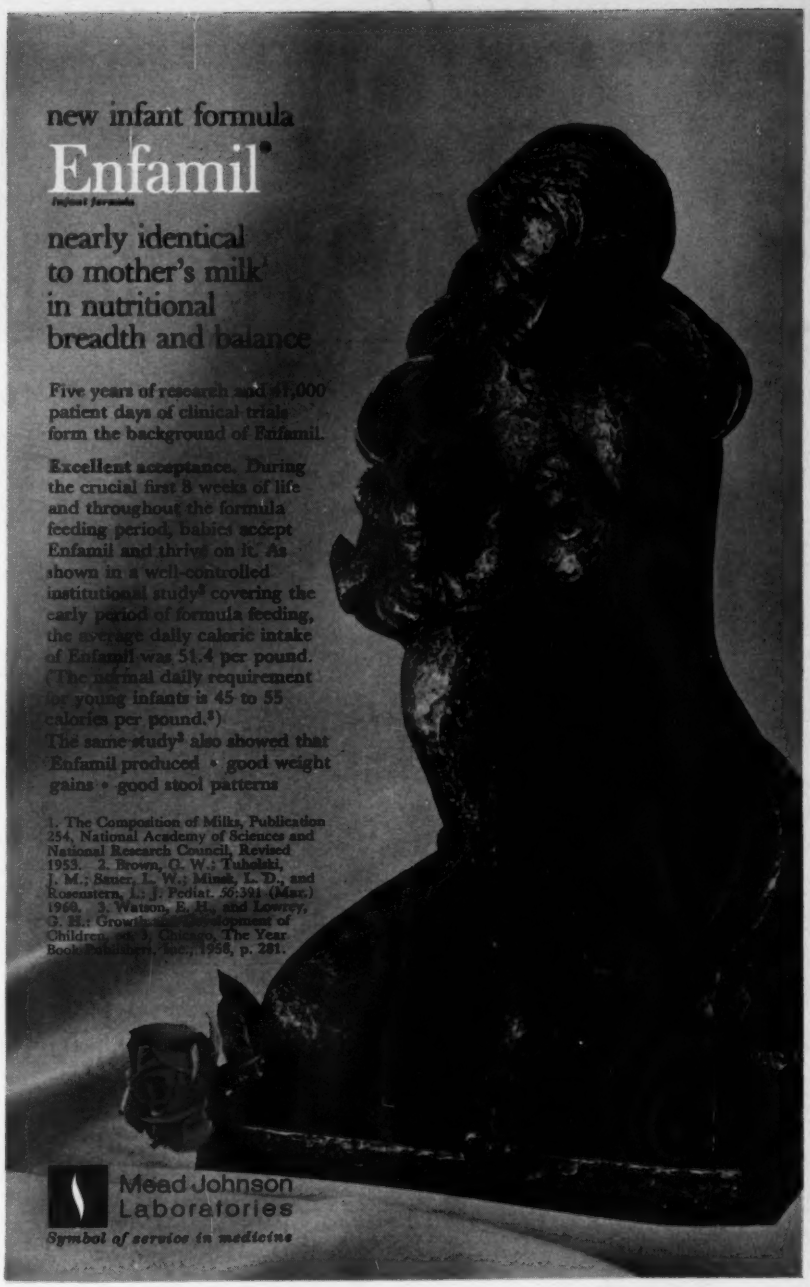
Address all communications to Paul van B. Allen, D.O., secretary, 4425 N. Meridian St., Indianapolis 8.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1. The Composition of Milks, Publication 254, National Academy of Sciences and National Research Council, Revised 1953. 2. Brown, G. W.; Tuholski, J. M.; Sauer, L. W.; Minsk, L. D., and Rosenstern, L.: J. Pediat. 56:391 (Mar.) 1960. 3. Watson, E. H., and Lowrey, G. H.: Growth and Development of Children, ed. 3, Chicago, The Year Book Publishers, Inc., 1956, p. 281.
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Causes of death in later life*

The very appreciable decreases in mortality among older people during recent decades have resulted in large part from the remarkable advances made in the control of the infectious diseases, particularly pneumonia and tuberculosis. This is evident from data collected which shows the trend of mortality since 1929-31 for the leading causes among white men and women in the age range 65-84 years.

Thanks to the antibiotics and other drugs, the death rate from pneumonia and influenza among white men at ages 65-84 decreased about two thirds between 1929-31 and 1958; among white women the corresponding reduction was as much as four fifths. Despite these gains, the respiratory diseases still are a serious health problem for older men and women, as evidenced by the increased mortality associated with these diseases in recent years.

Another dramatic feature of the mortality trend at the older ages has been the continuing decrease in the death rate from tuberculosis. Among white men at ages 65-74 the rate dropped 65 per cent between 1929-31 and 1958—from 123.1 to 43.0 per 100,000; at ages 75-84 the relative decrease was nearly as large. Among white women in the age range 65-84 the tuberculosis death rate fell more than 80 per cent.

Noteworthy progress has also been made in controlling the mortality from the cardiovascular-renal diseases in later life, particularly among women. The death rate from these causes among white women at ages 65-74 decreased 27 per cent in the period under review; even at ages 75-84 years the reduction was 16 per cent. Among white men at ages 65-74 the rate in 1958 was not appreciably different from that in 1929-31; it should be noted, however, that the trend was downward until the past few years. At ages 75-84, the death rate from the cardiovascular-renal conditions among men was somewhat lower in 1958 than at the beginning of the period, de-

*Reprinted from *Statistical Bulletin*, Metropolitan Life Insurance Company, October 1960.

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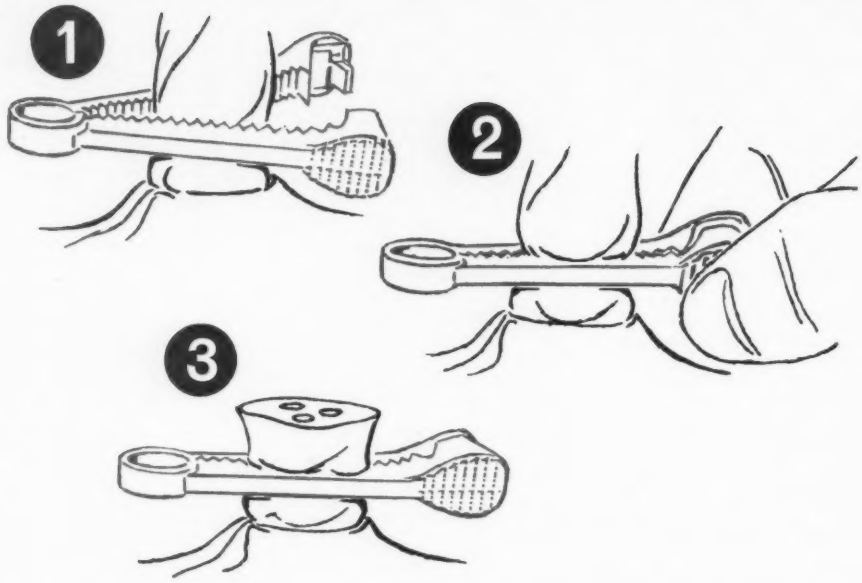

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spite the recent rise in mortality from these diseases. As a result of the more favorable trends for women, the excess in male mortality has increased markedly. For example, whereas the death rate at ages 65-74 was 21 per cent higher for males than for females in 1929-31, the excess was 68 per cent in 1958.

The mortality attributed to cancer (malignant neoplasms) showed contrasting trends for the two sexes. Since 1929-31, the death rate among white women decreased about 14 per cent at ages 65-74 and about 11 per cent at ages 75-84. On the other hand, the rate among white men increased approximately 27 per cent in these age groups. The adverse trend for the men reflects largely the marked rise in the recorded mortality from cancer of the respiratory system. At the older ages respiratory cancers are now responsible for 1 in every 5 deaths from malignancies among white men but for less than 1 in every 20 among the women.

Accident fatalities have decreased considerably in relative frequency among older men and women. For motor-vehicle accidents the decreases have been greater for men than for women, but for other accidents females have made the better record. The death rate from all accidents combined dropped nearly 50 per cent among white males in the age range 65-84, and almost 60 per cent among the women. By 1958, the excess of male over female mortality from accidents was as much as 90 per cent at ages 65-74 and 18 per cent at ages 75-84.

It is likely that further progress will be made in reducing mortality at the older ages. Because of their dominance in the current mortality picture, the cardiovascular-renal diseases are by far the greatest potential source of future gains. Even in the present state of knowledge, much more can be accomplished in postponing the onset of degenerative diseases, and in prolonging the life of patients with such conditions. Moreover, fuller use of specific means now at hand should make it possible also to reduce further the mortality from such causes as diabetes, accidents, pneumonia, tuberculosis, and other infectious diseases.

Consultation with parents of disturbed children*

David Hallowitz
Assistant Director
Albert V. Cutter, M.D.
Medical Director
and
Katharine Pitkin
Psychiatric Social Worker
The Psychiatric Clinic, Buffalo, N.Y.

For the past 8 years the Psychiatric Clinic in Buffalo, N.Y., has been treating some emotionally disturbed children with considerable success, entirely through consultation with their parents. This process of parent consultation takes place in one to five interviews with mother and father together. It is used with parents who are judged, in the initial interview, to have the ability to get at the roots of their part in the child's difficulties during the course of these interviews and, with little or no additional help, to go on to resolve the relationship problems within their family. Although the clinic routinely recommends psychiatric evaluation of each child as a safeguard and as an instrument for furthering the work with the parents, for the latter purpose, this evaluation is found to be unnecessary in about half the cases.

At the present time 18 per cent of the clinic's caseload falls in the parent consultation service classification. Without this service these cases would probably have been afforded full psychiatric treatment or have been on a long waiting list for it.

*Reprinted from *Children*, January-February 1961. Based on a paper presented at the 1960 meeting of the National Conference on Social Welfare.

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*Paul, W. D.: Rehabilitation in Rheumatoid Arthritis, South. M. J. *53*:492 (April) 1960.

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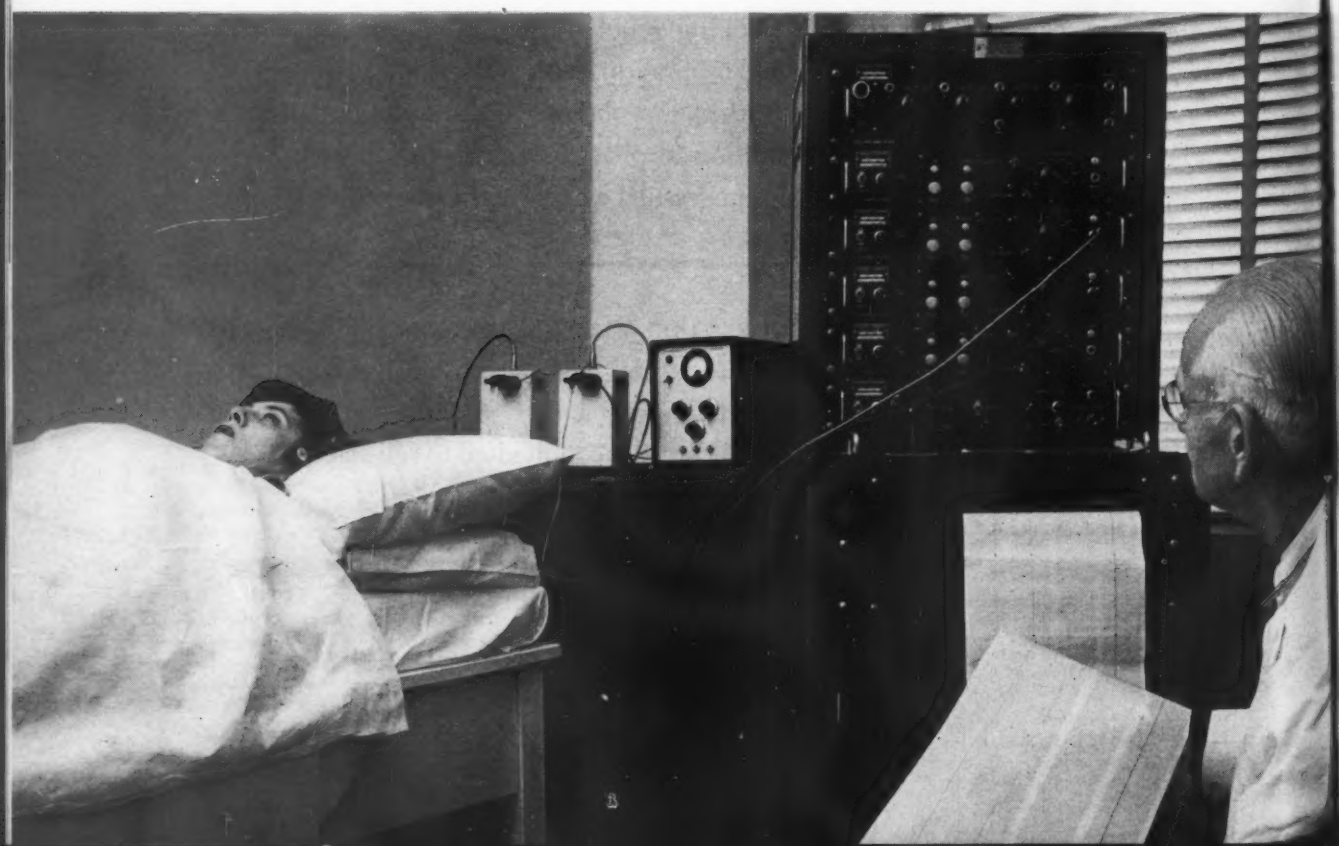

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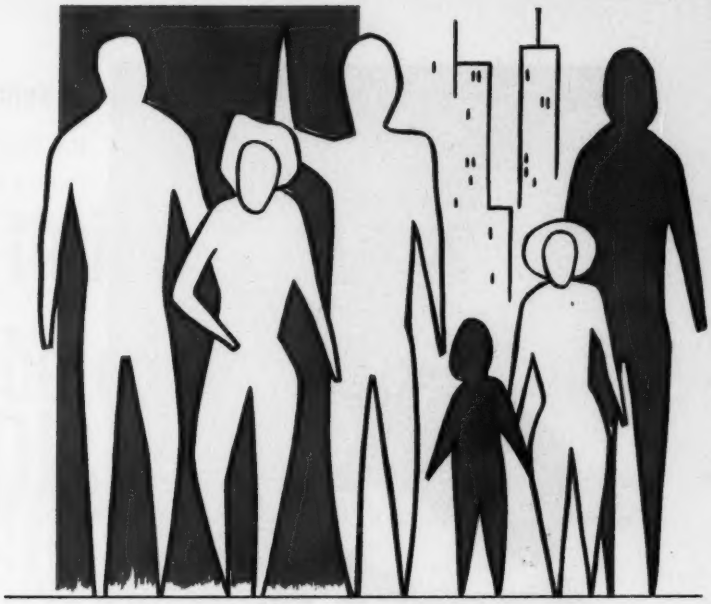

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exceptionally palatable liquid that children like to take. Each 5 ml. teaspoonful contains 5 mg. phenylephrine HCl and 2 mg. chlorprophenpyridamine maleate.

1. Beckman, H.: *Pharmacology: The Nature, Action and Use of Drugs*, 2nd Ed. W. B. Saunders Company, Philadelphia and London, 1961, p. 673.

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1. Wein, A. B.; The Use of Carisoprodol (SOMA) in Orthopedic Surgery and Rehabilitation, Miller, James G., ed., Wayne State University Press, Detroit, Michigan, 1959.

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the one nitrate for all with or without angina is Peritrate

Peritrate increases myocardial blood flow to the normal range and sustains it there[1]...without significant change in cardiac output,[1] blood pressure[2] or pulse rate.[3]

1. Johnson, P. C., and Sevelius, G.: J.A.M.A. 173:1231 (July 16) 1960. 2. Winsor, T., and Humphreys, P.: Angiology 3:1 (Feb.) 1952. 3. Plotz, M.: New York J. Med. 52:2012 (Aug. 15) 1952.

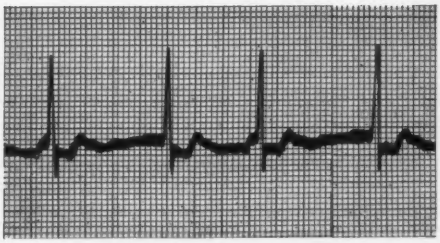
Full dosage information, available on request, should be consulted before initiating therapy.

*Electrocardiograms, radioisotopic tracings and case histories on file in the Medical Department of Warner-Chilcott Laboratories.

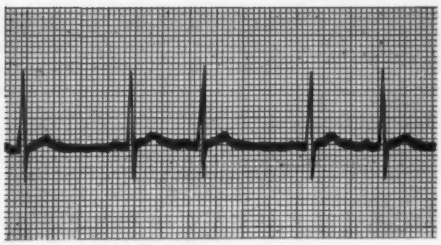
Electrocardiographic evidence:

Peritrate increases myocardial blood flow in a patient with angina*

The patient—tugboat captain, 57, with angina but no history of infarction. Blood pressure, 130/80. Normal sinus rhythm; ventricular rate, 72. Blood cholesterol, 344-583 mg./100 cc.



before **Peritrate**—S-T segment depressed after standard exercise.

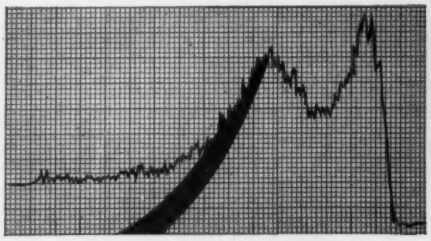


after **Peritrate**—(20 mg., given 4 hours before exercise test) S-T segment normal.

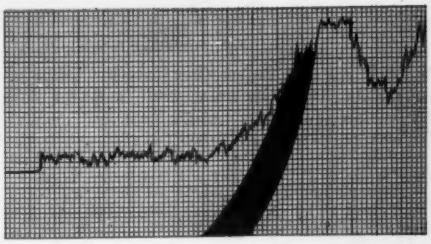
Radiocardiographic evidence:

Peritrate increases myocardial blood flow in a postcoronary patient without angina*

The patient—woman, 74, with 15-year history of hypertension. Posterior myocardial infarction in 1955. No angina. Before Peritrate: blood pressure, 210/110; pulse, 70. After Peritrate: blood pressure, 202/108; pulse, 68.



before **Peritrate**—Radioisotopic tracing shows myocardial blood flow (shaded area) after infarction reduced to 2.6% of cardiac output.



after **Peritrate**—(20 mg., given 2½ hours before study) myocardial blood flow increased to 5.9% of cardiac output.

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The essential features which differentiate parent consultation as used in the Psychiatric Clinic from other efforts to reach disturbed children through their parents[1-5] are:

1. It is effective for children of all ages.
2. The child's symptoms of disturbance are often multiple, of long duration, and bear a close resemblance to those of children requiring full diagnosis and direct psychiatric treatment.
3. The criteria for case selection and structure of the interviews are not applied in a preconceived manner, but are flexibly brought into play during the regular intake interview.
4. Mother and father (except when the mother is widowed or divorced) participate equally in a joint three-way interactive process with the worker.
5. The help given the parents is primarily in the realm of self-awareness and self-understanding.
6. Safeguards are rigorously instituted, and include followup procedures systematically applied over a period of several years.

Recently the clinic reviewed the 50 parent consultation cases which were active in 1954 and which were followed up regularly for from 2 to 5 years. Except for the stipulation that they be cases which were handled by senior staff members—to reduce as much as possible the variable of competence and skill—the cases were randomly selected. Twenty-eight—56 per cent—were regarded as successful; 8, or 16 per cent, partially successful, with residual problems; and 14, or 28 per cent, failures. Further service in the form of full diagnosis and treatment was offered in the 14 cases of failure, was declined by 9, and accepted by 5.

A case was considered to be successful if the child's presenting problems had cleared up following the help given the parents and if he seemed to be adjusting well within the family, in school, and with other children. Conversely, a case failure was defined as one in which the parents were not really helped and the child's problems did not become resolved. We realize that a margin for error exists in the objectivity and reliability of the parental versions of the child's progress or lack of progress but this, we think, would be minute.

In reviewing these 50 cases we not only compared the characteristics of the successes and the failures but also the characteristics of the total group with the characteristics of children in the treatment caseload as described in a previous study of 246 of our clinic cases.[6] For the sake of simplicity, we have combined the 36 successful and partially successful cases in making the comparison with the 14 failures. Here are some of the findings:

Age of child; sex; ordinal position; age of parents; socio-economic and educational status • No significant differences in these factors existed between the parent consultation cases and the total clinic caseload; nor in the association of these factors with success and failure in the two groups.

Presenting problems • A wide variety of symptoms of emotional disturbance is contained in the 50 parent consultation cases. Each child's symptomatic picture was crudely designated either as having an acting-out quality or a quality of internalization. The largest proportion of these children, 54 per cent—or a total of 27—were classified as "acting out," though among the failures as a group the reverse was true—55 per cent were regarded as having tendencies toward internalization.

Symptomatic acting-out behavior included such behavior patterns in the child as extreme defiance and control of parents; temper tantrums; belligerence; aggressive and destructive outbursts toward parents, siblings, teachers, and classmates; willful refusal to go to school or do schoolwork; stealing and other forms of delinquent behavior; fire setting. Symptoms of internalization tendencies included: enuresis and soiling; masturbation; stuttering; functional headaches and stomach aches; withdrawal and apathy; fears, nightmares, sleeplessness; obsessive-compulsive traits. While we recognized that particular symptoms

Athlete's foot is caused by fungi invading the horny, keratinized layers of the skin not reached by the normal blood supply. Topical application of DESENEX, a combination of zinc undecylenate and undecylenic acid, brings these powerful antifungal agents into direct contact with troublesome fungi and quickly relieves—**and arrests**—the annoying condition.

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eliminates the enema and straining at stool

In geriatric patients,[1,2] Peri-Colace promptly, yet gently, induces bowel movements—satisfactory evacuation without straining. It helps to eliminate the use of enemas for bedfast patients, even where fecal impaction is a complicating factor.[2]

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References: (1) Smigel, J. O.; Lowe, K. J.; Hosp, P. H., and Gibson, J. H.: M. Times *86*:1521-1526 (Dec.) 1958. (2) Napp, E. E., and Donnenfeld, A. M.: J. Am. Geriatrics Soc. *8*:858:860 (Nov.) 1960. (3) Broders, A. C., Jr.: Am. J. Digest. Dis. *2*:483-486 (Sept.) 1957.

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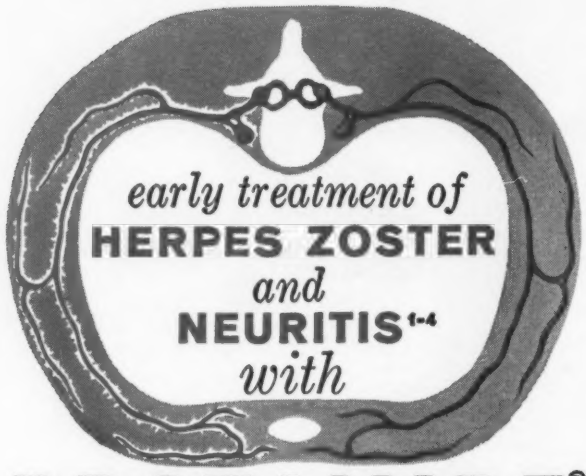

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References: 1. Baker, A. G.: Penn. Med. J. 63:697 (May) 1960. 2. Smith, R. T.: New York Med. (Aug. 20) 1952, pp. 16-19. 3. Smith, R. T.: Med. Clin. N. Amer. (Mar.) 1957. 4. Lehrer, H. W.; Lehrer, H. G., and Lehrer, D. R.: Northw. Med. (Nov.) 1955. 5. Sforzolini, G. S.: Arch. Ophthal. 62:381 (Sept.) 1959.

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Detroit 11, Michigan

might relate to either tendency, we tried to assess them in relation to the child and the family situation as a whole.

Our purpose in this rather unscientific examination of symptomatic behavior was to find out whether or not the children of the parent consultation cases resembled those who needed full diagnosis and treatment. Symptomatically they did resemble the other children, for the percentage of acting-out children in the total caseload, 51 per cent, was only slightly lower than in the parent consultation group. However, among the children of the parent consultation cases, symptoms suggesting neurosis or perpsychosis proved to be either signs of the very early stages of the condition or completely superficial and illusory, and tended to disappear in a relatively short period of time—a few weeks to a few months—while the child became better adjusted in every area. The conclusion that the children in parent consultation cases were not nearly so disturbed as the children in the treatment caseload was borne out by the psychiatric evaluations that have been carried out on the former during the past 2 years.

Prior duration of child's difficulties • Among the parent consultation cases the length of time a child had exhibited difficulties before the parents came to the clinic varied widely, ranging from 4 or 5 months to 8 or 9 years. But 37 per cent of the successful cases had a prior-problem-duration of less than 2 years, compared with 25 per cent of the failures.

The prior duration of the child's symptoms was generally shorter in the parent consultation cases than in the regular treatment cases. It was also shorter in the successful cases than in the unsuccessful ones, but not substantially so.

Parent problem areas • Here, the only comparison made was between the successful cases and the failures. The parental problems in the successful cases were markedly less complicated and severe than in the unsuccessful ones. The parents' problem in 92 per cent of the successful cases existed solely in their relationship to the child. However, in 65 per cent of the cases of failure the problem was complicated by marital disturbances or mental illness in one of the parents. An additional factor in the group of failures was a deeper disturbance in some of the children than was recognized in the initial interview.

CRITERIA FOR SELECTION

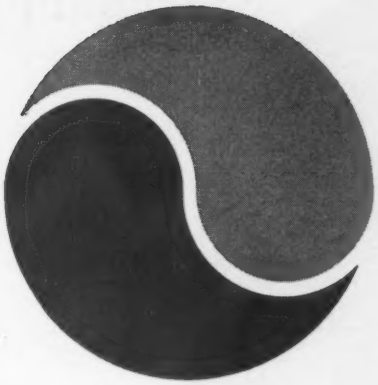
On the basis of these comparisons, it is possible to formulate criteria of a case suitable for parent consultation. These criteria cannot be applied inflexibly but only as working guides. Exceptions can be made.

Child • The child's symptoms, even though they may suggest the beginnings of a neurotic or prepsychotic process, are not yet deeply ingrained but are principally reactions to weakness, conflict, and breakdowns in the parent-child and parent-parent (re child) relationships. In fact, the child gives evidence of having substantial areas of inner strength.

The child's age and the prior duration of his symptoms are not determining factors in the choice of the parent consultation approach. The approach has proved effective with adolescents as well as with preschool children. It has also been effective in cases in which the child's symptoms have been present for several years. However, the younger the child and the shorter the duration of the symptoms, the better are the chances of a successful outcome.

Parents • The parents are in basically good emotional health as individuals, and their marital relationship is fairly sound. They genuinely wish to examine themselves and their part in the child's emotional difficulties. Moreover, they are capable of doing this securely and with a minimum of resistance and defensiveness. They are quick to gain insights, grasp concepts—on an emotional as well as in intellectual level—and to apply their new understanding with conviction.

Although the parents feel frustrated and angry with the child and themselves, on a deeper level they love him dearly.[7] The breakdowns are in the superstructure



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SUPPLIED: Tablets, bottles of 100. Each tablet contains 25 mg. of HYDRODIURIL (hydrochlorothiazide) and 200 mg. of meprobamate.

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of intrafamilial relationships. The foundation is a good one upon which the relationships can be repaired or reconstructed.

The age of the parents and their socioeconomic and educational level are not determining factors. However, the clinical impression is that younger parents (parents in their twenties and thirties) are more resilient and amenable to change than parents in their forties and fifties. Good intelligence—but not necessarily higher education and social position—is a favoring factor.

THE DYNAMICS

Let us turn now to the dynamics of the parent consultation process. In the 90-minute joint intake interview with the parents the worker arrives at presumptive diagnostic impressions about the child through evaluation of his symptoms, the origins of their development, and the significant medical and developmental history. Through discussion, and direct observation of the parents and their interaction with each other, the worker also gains diagnostic impressions of the parent-child relationships, the parent-parent relationship, and the state of each parent's emotional health.

In about the middle of this session, the worker makes the hypothetical determination as to whether or not the family situation approximates the criteria we have described. Instead of the traditional detailed history-gathering and extensive discussion of parental feeling about seeking help, he focuses upon the parents' problems which might underlie the child's difficulties. He tries to help them realize that in spite of their problems with the child and angry feelings toward him, and perhaps toward each other, they are not "bad" parents but only lost in their parenting and have many good, healthy qualities on which to build. By thus reviving and reinforcing their own self-confidence, the worker helps the parents to achieve understanding on an emotional level of the ways in which they have contributed to the child's problems. The worker helps the parents to formulate plans of action themselves, but also gives specific directions and suggestions in a supportive manner.

Mr. and Mrs. S described Larry, age 11, as always having been rather quiet, introverted, a worry wart, and a perfectionist, but said they had not become alarmed about the boy until 8 or 9 months before they came to the clinic. They said the child's neatness seemed to be almost an obsession with him. He arranged shoes in his closet, smoothed out his clothes, made numerous minute adjustments of his music stand, straightened and line up papers, closed all desk and bureau drawers carefully. They said that Larry had never been a disciplinary problem and had always been well liked, but now seemed to have reached a point where he had no friends and apparently no desire for any. They reported that Larry's teacher liked him, but that her patience had become exhausted with his puttering and slowness.

Larry's developmental and medical history was normal. The social worker had previously learned from the pediatrician[8] that no medical or organic problems were present, and from the school that the boy was of average intelligence and performed adequately prior to the intensification of his difficulties during the past year. In a brief discussion with the parents the worker also learned that their marital relationship and earlier family background seemed to be normal.

With these indications that a parent consultation process might be applicable, the social worker now began to focus upon the underlying problems in the parent-child relationships, and in the parents' relationship with each other as this affected Larry.

The social worker asked the parents what they thought might explain the child's symptoms. Mrs. S tentatively suggested that perhaps she and her husband had not been spending enough time with Larry. Mr. S added that he did not have a close relationship with the child. He said he had always found it difficult to get the boy to do things with him. He said, for example, that if he were washing the car, Larry would not come over to help, whereas Nancy,

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1. Schwartz, H.A.: Editorial, Minnesota Med. 42:1279, 1959.

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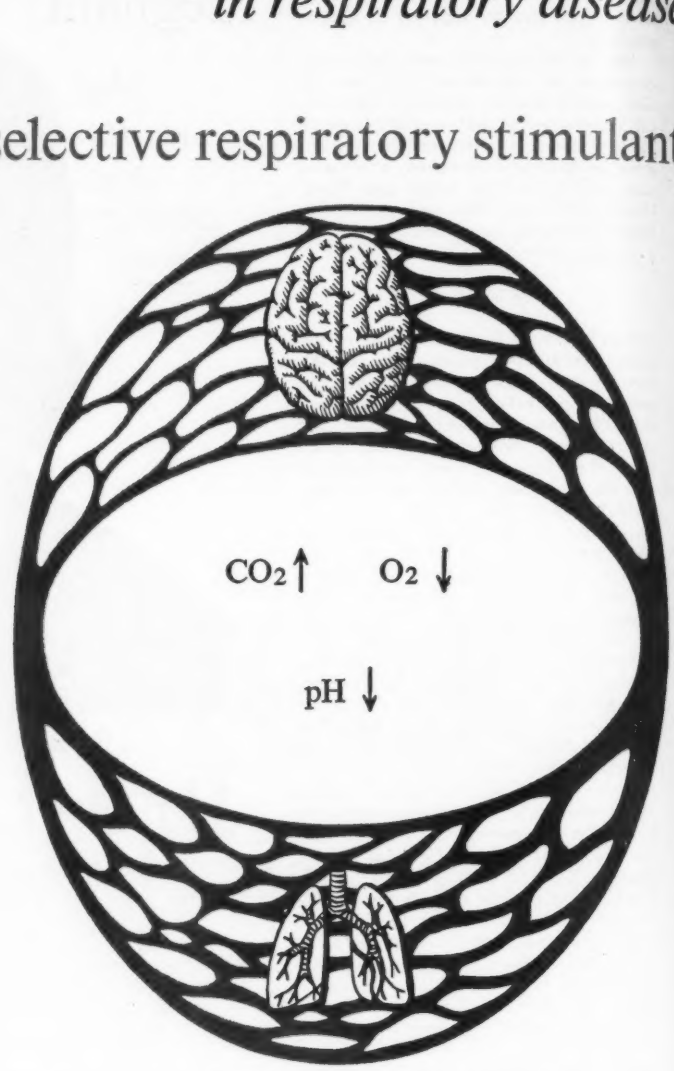

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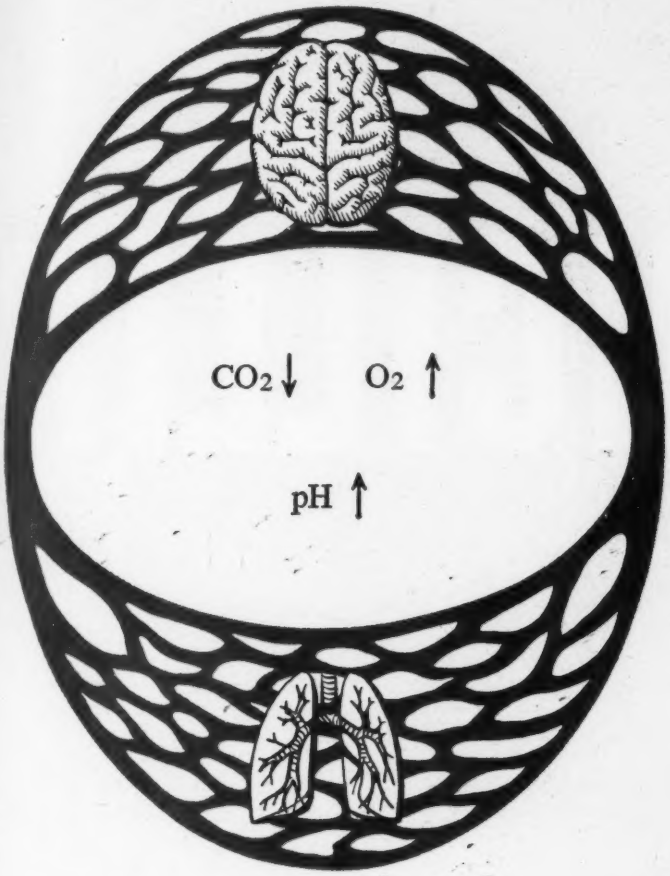

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affirms the clinical significance of its unique property of virtually withstanding destruction by staphylococcal penicillinases

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affirms that Staphcillin is free from such toxic hazards as hearing impairment, renal damage, and blood dyscrasias characteristic of other antibiotics active against resistant staphylococci

BRISTOL

STAPHCILLIN...EFFECTIVE IN LIFE-THREATENING INFECTIONS CAUSED BY RESISTANT STAPHYLOCOCCI

Endocarditis, Septicemia, and Osteomyelitis

A 10-year-old boy was admitted to the hospital on August 11, 1960, acutely ill with high fever, and severe pain and swelling of the right knee, right thigh, and right elbow. Six days before, he had injured his right knee. On admission he had a systolic murmur. A short time later cardiac enlargement, slurring of speech, and ataxia of the right arm and leg developed. Blood culture revealed an abundant growth of coagulase-positive staphylococci; the same organisms were later identified in material drained from a large abscess surrounding part of the right femur, which was denuded of periosteum. The diagnosis was acute osteomyelitis, septicemia, and endocarditis caused by staphylococcal infection.

Over a period of 7 weeks, the following antibiotics were administered, but were discontinued because of inadequate improvement or toxicity: penicillin G, tetracycline, chloramphenicol, kanamycin, vancomycin, novobiocin, erythromycin, and ristocetin. Staphcillin was started on Sept. 29, 1960, 1 Gm. I.M., q. 6 h. (later reduced to 3 Gm. daily), and was given for 54 days. Shortly following initiation of this therapy, he became afebrile, and there was marked clinical improvement thereafter. There were no side effects from Staphcillin. As of March, 1961, he had had no recurrence of the infection, was afebrile, had gained weight and the ataxia and speech defect had almost disappeared. There was residual heart murmur. There were signs of osteomyelitis in the right femur on X-ray examination.

Evans, W. E.: Report to Dept. of Clin. Invest., Bristol Laboratories.

Empyema and Concurrent Pseudomonas Wound Infection

A 61-year-old male was injured in an automobile accident. He was operated upon for lacerations of the spleen, left diaphragm, pericardium, and left ventricle. He later developed a *Staph. aureus* empyema and a *Pseudomonas aeruginosa* abdominal wound infection. Chloramphenicol, erythromycin, and penicillin G were given, but discontinued because of inefficacy. Staphcillin was then administered, 2 Gm. q.i.d., for 30 days. Polymyxin B was administered concurrently for the Pseudomonas infection. The condition was improved, with apparent elimination of all infecting bacteria.

Hewitt, W. L.: Report to Dept. of Clin. Invest., Bristol Laboratories.

Pneumonia following Laparotomy

A 37-year-old man had a laparotomy performed at which acute pancreatitis was found. Postoperatively, he developed a bilateral staphylococcal bronchopneumonia. The organism was resistant to penicillin G. Before the laboratory tests were reported, he had been given massive doses of penicillin G for three days with no temperature response. Staphcillin was given intravenously, 1 Gm. every four hours, after which his temperature became normal in 48 hours, "and his clinical response was most gratifying."

Branch, A.; Rodger, K. C.; Lea, R. W., and Power, E. E.: Canad. M. A. J. 83:991 (Nov. 5) 1960.

Enterocolitis after Abdominal Surgery

A 65-year-old woman had profuse watery diarrhea 3 days after an abdominal operation. She became acutely ill, with a temperature of 103°F., tachycardia, leukocytosis, and fluid and electrolyte loss. Staphylococci in pure culture were identified on stool smear and culture. Tetracycline, which she had been receiving since operation, was discontinued, and Staphcillin (1.5 Gm. every 6 hours) administered parenterally. Within 48 hours she was afebrile, and the diarrhea had subsided. Staphylococci were eliminated from the stool. She received Staphcillin for 8 days, and was discharged as well on the 11th postoperative day.

Rutenburg, A. M.; Greenberg, H. L., and Schweinburg, F. B.: New Eng. J. Med. 263:1174 (Dec. 8) 1960.

Septicemia and Empyema — Abscess on Hip

A 4-week-old infant was gravely ill with a large abscess on the right hip. *Staph. aureus*, type 80, was isolated from this abscess, from blood culture and urine, and from an empyema. There was no response to parenteral penicillin G and streptomycin, and treatment was begun with sodium methicillin. There was immediate improvement; the blood culture and urine became sterile; and within 5 days the discharge from the abscess cavity and empyema were also sterile. The infant's general condition improved, and within two weeks he was lively and feeding well. Treatment was continued for 20 days, during which time all signs of active infection subsided. "There is little doubt," reported the investigators, "that in this case a severe staphylococcal pyema was rapidly controlled and that BRL 1241 [sodium methicillin] had a life-saving effect."

Stewart, G. T.; Nixon, H. H., and Coles, H. M. T.: Brit. M. J. II:703 (Sept.) 1960.

STAPHCILLIN...EFFECTIVE IN SKIN, SOFT TISSUE AND BONE INFECTIONS CAUSED BY RESISTANT STAPHYLOCOCCI

Persistent Multiple Boils

A 58-year-old male, who for many weeks had suffered from multiple boils from which resistant staphylococci were isolated, was treated for 3 days with sodium methicillin, 1 Gm., q. 4 h., followed by 1 Gm. q. 6 h. for another 3 days. Previous treatment with chloramphenicol and erythromycin had been unsuccessful. At the end of the 6-day period there was complete resolution of all the boils, and the patient was discharged from the hospital. There has been no recurrence.

Knudsen, E. T., and Rollnson, G. N.: Brit. M. J. II:700 (Sept.) 1960.

Cellulitis and Abscess

A 22-year-old male was treated for cellulitis and purulent abscess of the left thigh, resulting from a wound. *Staph. aureus*, resistant to penicillin G, was isolated from the wound tissue. Penicillin G, tetracycline, and erythromycin proved ineffective. Staphcillin was then administered for 17 days, 1 Gm., 5 times daily. The elimination of the infective organisms was impressive.

Bunn, P. A.: Report to Dept. of Clin. Invest., Bristol Laboratories.

Submandibular Abscess

A 24-year-old man was suffering from a submandibular abscess secondary to obstruction and infection in the submandibular duct. This abscess was drained surgically three days before antibiotic therapy, and a heavy growth of coagulase-positive *Staph. aureus* was cultured from the wound. He was given chloramphenicol (to which the organism was reported sensitive) for 7 days, but continued to have a positive culture. Staphcillin was then given intramuscularly for one week. The temperature dropped from 99.6°F. to normal within 24 hours. The drainage soon disappeared. The patient recovered, and was discharged from the hospital two weeks later without any subsequent antibiotic treatment.

Branch, A.; Rodger, K. C.; Lee, R. W., and Power, E. E.: Canad. M. A. J. 83:991 (Nov. 5) 1960.

Osteomyelitis of Femur — Recurrent for 3 Years

A 42-year-old woman had a recurrent osteomyelitis of the left femoral shaft for three years. Upon this admission to the hospital, swelling, heat and tenderness of the thigh were present. Operation revealed a purulent abscess extending into the medullary bone. Cultures showed *Staph. aureus*. The patient was treated with Staphcillin and probenecid for 6 days, following which the drainage became temporarily negative for staphylococci. After an interval of 10 days, a second course of the same treatment was started. Eight days later, the drainage became negative for staphylococci, and subsequently the incisions closed. The patient was treated for a total of 35 days, and was discharged as symptomatically improved. She was readmitted two months later, and was again treated with 8 Gm. of Staphcillin and 1½ Gm. of probenecid per day, for three months, with recovery.

Rifkind, D., and Knight, V.: Report to Dept. of Clin. Invest., Bristol Laboratories.

Retropharyngeal Abscess

A 24-year-old male was treated for a prevertebral, retropharyngeal abscess. The infection was caused by *Staph. aureus*, phage type 80/81. Tetracycline, novobiocin, and vancomycin were only partially effective. Staphcillin was then given I.M., 1 Gm. 6 times per day, for 24 days. The patient was cured. There was a mild local reaction, at the site of injection.

Thompson, R. A.: Report to Dept. of Clin. Invest., Bristol Laboratories.

Multiple Carbuncles and Furunculosis

A 53-year-old male orderly with multiple carbuncles and furuncles, from which staphylococci resistant to penicillin G were cultured, had been followed as an outpatient for five weeks. New lesions occurred in different areas, from which the same organisms were isolated. Staphcillin was given intramuscularly, 1 Gm. q. 6 h., for seven days. Follow-up cultures were negative, and all lesions were closed.

Branch, A.; Rodger, K. C.; Lee, R. W., and Power, E. E.: Canad. M. A. J. 83:991 (Nov. 5) 1960.

Vertebral Osteomyelitis with Retroperitoneal Abscess (Superinfection)

An 18-year-old patient with recurrent staphylococcal osteomyelitis of the spine, developed a retroperitoneal abscess. Previous therapy with chloramphenicol and erythromycin was ineffective. Staphcillin was given, 2 Gm. q. 6 h., for 18 days, and 1 Gm. q. 6 h., for 17 days. Superinfection with *Pseudomonas aeruginosa* occurred, and polymyxin B was given concurrently with Staphcillin. There was excellent clinical and bacteriological response to both antibiotics. Five months later, neither infection had recurred.

Hewitt, W. L.: Report to Dept. of Clin. Invest., Bristol Laboratories.

STAPHCILLIN THERAPY...

Summary of response in staphylococcal disease

TYPE OF INFECTION	NO. OF PATIENTS	S*	N.C.	I	D
Soft tissue infection	187	158	7	14	8 (2)
Lower respiratory (pneumonia, empyema bronchitis, lung abscess)	70	49	5	4	12 (3)
Osteomyelitis	54	43	6	4	1 (0)
Septicemia	41	30	3	2	6 (1)
Endocarditis (acute & subacute)	25	22	0	1	2 (0)
Genitourinary	17	14	1	1	1 (0)
Upper respiratory	15	13	2	0	0 (0)
Enterocolitis	12	12	0	0	0 (0)
CNS (abscesses and meningitis)	6	3	1	0	2 (1)
Septic arthritis	6	5	0	0	1 (0)
	433	349	25	26	33 (7)

*[S-satisfactory response; improved or cured] [NC-no change] [I-indeterminate response; not stated or insufficient data] [D-died; ()-No. of deaths due to infection; other deaths were due to causes such as coronary thrombosis, pulmonary embolism, terminal carcinoma, etc.]

The data in the above table were compiled from published articles and reports to The Department of Clinical Investigation of Bristol Laboratories.

Of the 433 patients included in this summary, 256 received one or more of 21 different antibacterial agents prior to initiation of Staphcillin therapy. Among these patients, 147 received chloramphenicol, 72 received erythromycin, 34 received kanamycin, 28 received novobiocin, 28 received vancomycin.

The two major reasons given for discontinuing other antibiotics were:
1. previous antibiotic was ineffective; 2. previous antibiotic demonstrated toxicity.

SUMMARY OF SENSITIVITY STUDIES

Published data and reports to the Department of Clinical Investigation, Bristol Laboratories, concerning sensitivity of staphylococci to Staphcillin are summarized to date (March, 1961) as follows:

Total number of isolates 8,060

No. of isolates with a Minimum Inhibitory Concentration (M.I.C.) of 6.25 mcg./ml. or less (45 investigators) 8,052

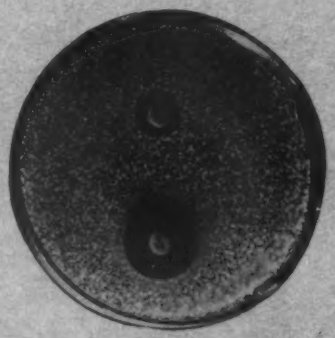
No. of isolates with an M.I.C. of more than 6.25 mcg./ml. (4 investigators: Jevons, Rutenburg, Katz, Eichenwald).... 8 (0.09%)

Blood levels well over 12 mcg./ml. are commonly reached for limited periods following the recommended I.M. or I.V. dosage of Staphcillin. Many infections caused by strains with an M.I.C. of 6.25 mcg./ml. have responded well to treatment with the recommended dosage. The rare incidence to date of strains of staphylococci with an M.I.C. of more than 6.25 mcg./ml. is shown in the above summary (0.09% of 8,060 isolates). Only in the future, following treatment of a sufficient number of such cases, can it be known whether they will respond to Staphcillin, and further, what the highest M.I.C. might be that is consistent with good clinical response.

THE ACTIVITY OF STAPHCILLIN IS VIRTUALLY UNAFFECTED BY STAPHYLOCOCCAL PENICILLINASES

The width of the clear zones around the sensitivity discs in Figs. 1 and 2 shows that Staphcillin acts against this resistant strain, and that penicillin G has little or no effect. The similarity in the width of the zones around Staphcillin discs in the light and heavy (increased concentration of penicillinase) inocula shows that penicillinase-producing staphylococci have virtually no effect on the antistaphylococcal activity of Staphcillin.

The clinical effectiveness of Staphcillin against penicillin G-resistant staphylococci is most probably related to Staphcillin's demonstrated resistance to destruction by penicillinase.



Penicillin G

Fig. 1
Light Inoculum

STAPHCILLIN

Penicillin G

Fig. 2
Heavy Inoculum

STAPHCILLIN

CONCURRENT ANTIBIOTIC THERAPY IN MIXED OR SUPERINFECTIONS

If mixed or superinfections due to Gram-negative organisms occur during Staphcillin therapy, appropriate measures should be taken. Clinical experience reported to the Department of Clinical Investigation, Bristol Laboratories can be summarized as follows: One or more antibiotics were employed concurrently with Staphcillin in the treatment of 88 patients. Of these, 73 improved, and there was no change in 5. There were 10 deaths, all of which were unrelated to antibiotic therapy; e.g., coronary thrombosis, pulmonary embolism, terminal carcinoma, etc. Among the antibiotics most commonly used with Staphcillin in this series of 88 patients were polymyxin, colistin, bacitracin, streptomycin, kanamycin and chloramphenicol.

STAPHCILLIN®

sodium methicillin (sodium dimethoxyphenyl penicillin)

FOR INJECTION

INDICATIONS: Staphcillin is recommended specifically for infections caused by strains of staphylococci susceptible to it and resistant to other penicillins. When laboratory sensitivity tests suggest that the staphylococci involved in an infection are susceptible to Staphcillin and to other antimicrobial agents, selection can be further guided by judgment of the nature of the infection, of the need for bactericidal action, and by considerations of comparative toxicity and convenience of administration.

SUCH INFECTIONS INCLUDE:

Skin and soft tissue infections: cellulitis, wound infections, carbuncles, pyoderma, furunculosis, lymphangitis and lymphadenitis.

Respiratory infections: staphylococcal lobar pneumonia, bronchopneumonia, or lung abscess combined with indicated surgical treatment.

Other infections: staphylococcal septicemia, bacteremia, acute or subacute endocarditis, acute osteomyelitis and enterocolitis.

Infections due to penicillin-sensitive staphylococci, streptococci, pneumococci or gonococci, should be treated with Syncillin (potassium phenethicillin) or parenteral penicillin G, rather than with Staphcillin. Treponemal infections should be treated with parenteral penicillin G.

DOSAGE AND ADMINISTRATION:

Intramuscularly (deep intragluteal): Adults — The usual dose is 1 Gm. every 4 to 6 hours. Infants and children — 25 mg. per kg. of body weight (approximately 12 mg. per lb.) every 6 hours.

Intravenously: Adults — The usual dose is 1 Gm. every 6 hours, using 50 ml. of sterile saline solution at the rate of 10 ml. per minute.

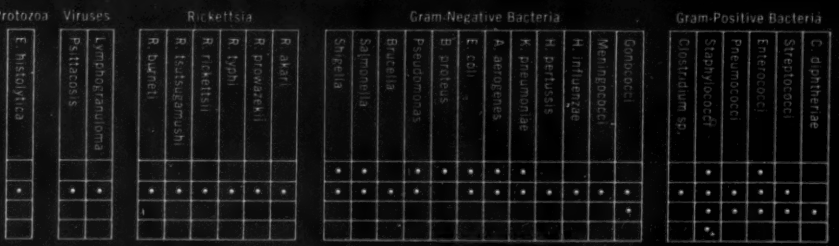
PRECAUTIONS: Mild skin reactions, e.g., itching, papular eruptions, and erythema have occasionally been observed during treatment and after discontinuance of therapy. Patients with histories of hay fever, asthma, urticaria, and previous sensitivity to any penicillin are more likely to react to all penicillins, and the possibility of anaphylaxis should be kept in mind; epinephrine, antihistamines, and intravenous corticosteroids should be available for emergency treatment. Because of the virtually complete resistance of Staphcillin to destruction by penicillinase, parenteral *B. cereus* penicillinase may not be effective for treatment of allergic reactions. If superinfection by Gram-negative organisms or fungi occurs during Staphcillin therapy, appropriate measures should be taken.

SUPPLY: Staphcillin is available in 1 Gm. dry-filled vials, equivalent to 900 mg. of dimethoxyphenyl penicillin activity.

References: 1. Knox, R.: Brit. M. J. ii:690 (Sept. 3) 1960. 2. Stewart, G. T., *et al.*: Brit. M. J. ii:694 (Sept. 3) 1960. 3. Robinson, G. N., *et al.*: Lancet ii:564 (Sept. 10) 1960. 4. Rutenburg, A. M., *et al.*: New Eng. J. Med. 263:1174 (Dec. 8) 1960. 5. Bunn, P. A., *et al.*: New York J. Med. 60:3074 (Oct. 1) 1960. 6. Branch, A., *et al.*: Canad. M. A. J. 83:991 (Nov. 5) 1960. 7. Gourevitch, A., *et al.*: Paper presented at Conference on Staphcillin in the Therapy of Resistant Staphylococcal Disease, Sept. 7, 1960, State University of New York College of Medicine, Syracuse, New York. 8. Casson, K., and Lannon, J.: *vide supra*. 9. White, A. C., and Varga, D. T., *vide supra*. 10. Knight, V., and Rifkind, D., *vide supra*. 11. Douthwaite, A. H., and Trafford, J. A. P.: Brit. M. J. ii:687 (Sept. 3) 1960. 12. Pindell, M. H., *et al.*, *vide supra*. 13. Smith, I. M., and Counts, G. W., *vide supra*. 14. Hewitt, W. L., *et al.*, *vide supra*.

PROFESSIONAL INFORMATION SERVICE: Whenever you desire additional information concerning clinical experience with Bristol antibiotics, you may avail yourself of the services of the Medical Department of Bristol Laboratories via a collect telephone call to New York, PLaza 7-8520, or by mail to the Medical Department, Bristol Laboratories, 630 Fifth Avenue, New York 20, N. Y.

SPECTRUM CHART OF BRISTOL ANTIBIOTICS



(A-kanamycin sulfate) (B-tetracycline phosphate complex) (C-potassium phenethicillin) (D-sodium methicillin) Since not all strains of the microorganisms listed may be sensitive to a particular antibiotic, clinical therapy should be guided by in vitro antibiotic sensitivity tests whenever possible. *recommended for those staphylococci resistant to other penicillins

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are opiates now outmoded in pediatric diarrhea?

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the first pharmacologically-specific, non-narcotic antiperistaltic agent ■ controls diarrhea as rapidly and effectively as opiates ■ without the undesirable properties of opiates ■ pleasant butterscotch flavor

AND WHEN THE DIARRHEA IS BACTERIAL IN ORIGIN

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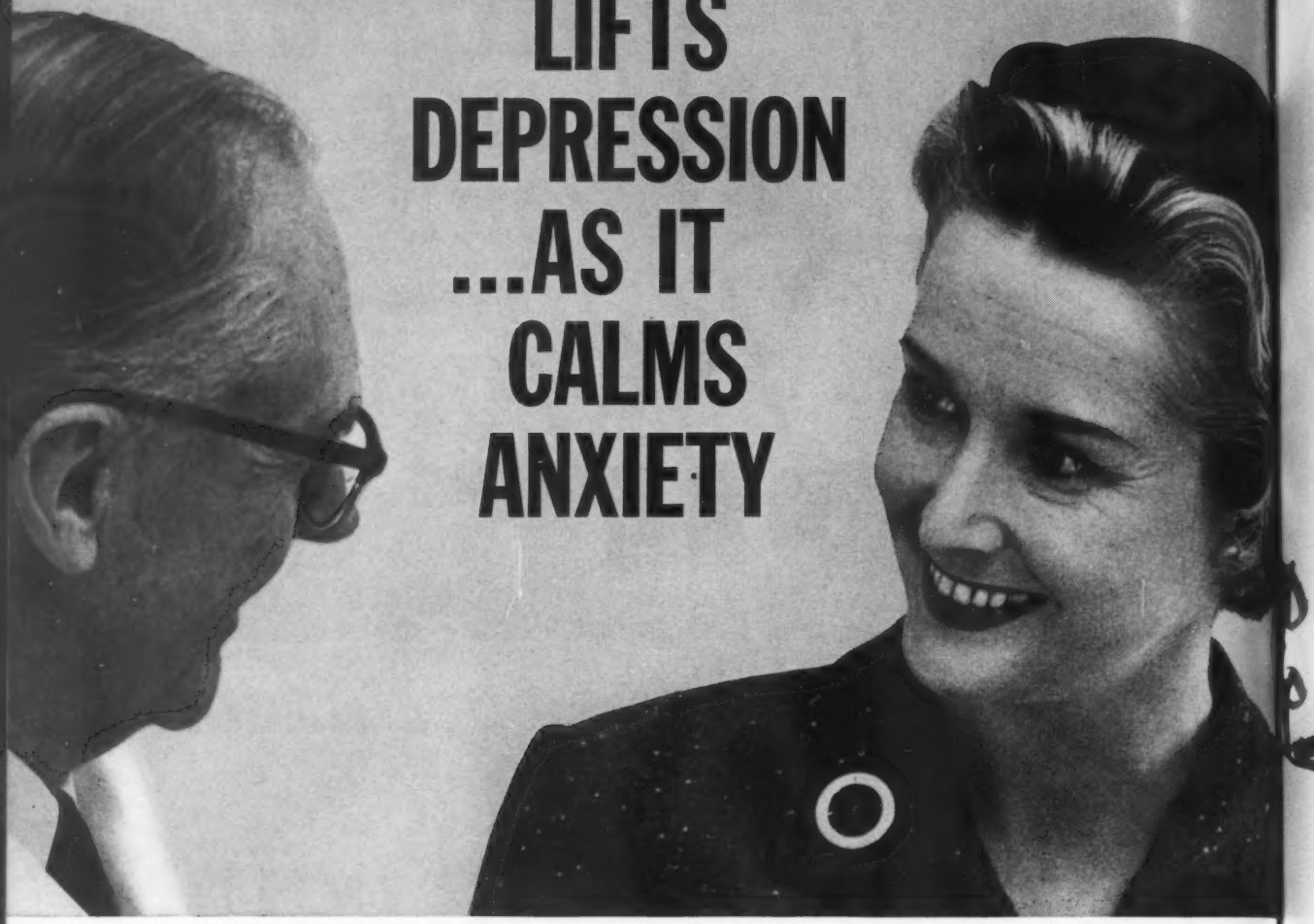
LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

LIFTS DEPRESSION ...AS IT CALMS ANXIETY



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Brightens up the mood, brings down tension

Deprol's balanced action avoids "seesaw" effects of energizers and amphetamines. While energizers and amphetamines may stimulate the patient – *they often aggravate anxiety and tension.*

And although amphetamine-barbiturate combinations may counteract excessive stimulation – *they often deepen depression and emotional fatigue.*

These "seesaw" effects are avoided with Deprol. It lifts depression as it calms anxiety – a balanced action that brightens up the mood, brings down tension, and relieves insomnia, anorexia and emotional fatigue.

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Dosage: Usual starting dose is 1 tablet q.i.d. When necessary, this dose may be gradually increased up to 3 tablets q.i.d.
Composition: 1 mg. 2-diethylaminoethyl benzilate hydrochloride (benactyzine HCl) and 400 mg. meprobamate.
Supplied: Bottles of 50 light-pink, scored tablets.
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for infants allergic to cow's milk

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Sobee has the rich, creamy appearance that mothers expect of a formula. Sobee is pleasantly bland, without the "burned-bean" flavor or chalky aftertaste frequently associated with a soya formula.

Symptomatic Relief. Symptoms of cow's milk allergy—most frequently manifested by eczema, colic and gastrointestinal disturbances—may be relieved within 2 or 3 days.

Good Stool Pattern. In a study of 102 infants on Sobee, the number of stools ranged from 1 to 4 per day.[1] Soya stools are bulkier than cow's milk stools. Constipation is infrequent.

Easily Prepared. Mothers need add only water to either Sobee liquid or Sobee instant powder to prepare a formula with a nutritional balance comparable to cow's milk formulas.

1. Kane, S.: Am. Pract. & Digest Treat. *8*:65 (Jan.) 1957.

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age 6, would eagerly do so. Mr. S recalled that when Larry was a young child, he would become discouraged when Larry would not come with him to take an automobile ride. When the worker encouraged him to talk about his feelings in regard to this, he said he used to feel angry and hurt but lately had come to feel badly about himself, thinking that he must have something missing which prevented him from having a closer relationship with his son. Smiling sheepishly, he went on to say that he used to feel jealous toward his wife because she was the one who had the close relationship with Larry. When the worker commented that he probably also felt resentment, he then admitted that he did, and turned to his wife, saying: "Whenever I am at the point when I want to talk with Larry about something, you are always right there ahead of me."

This kind of discussion led the parents to become aware of how Mrs. S unwittingly got in the way of her husband's efforts to reach Larry. She herself had been feeling resentful toward her husband for not having a closer relationship with Larry, thereby leaving the burden of this to her, and had thought that she had to make up for her husband's weakness in this respect. This was a revelation to Mr. S.

Explaining briefly a boy's early attachment to his mother as part of a normal oedipal struggle, the social worker commented on how difficult the father's pulling away from Larry must have made it for the child to come through this stage of his emotional growth. The worker pointed out that in spite of his apparent rejection of his father, Larry really has a great need for a relationship with him.

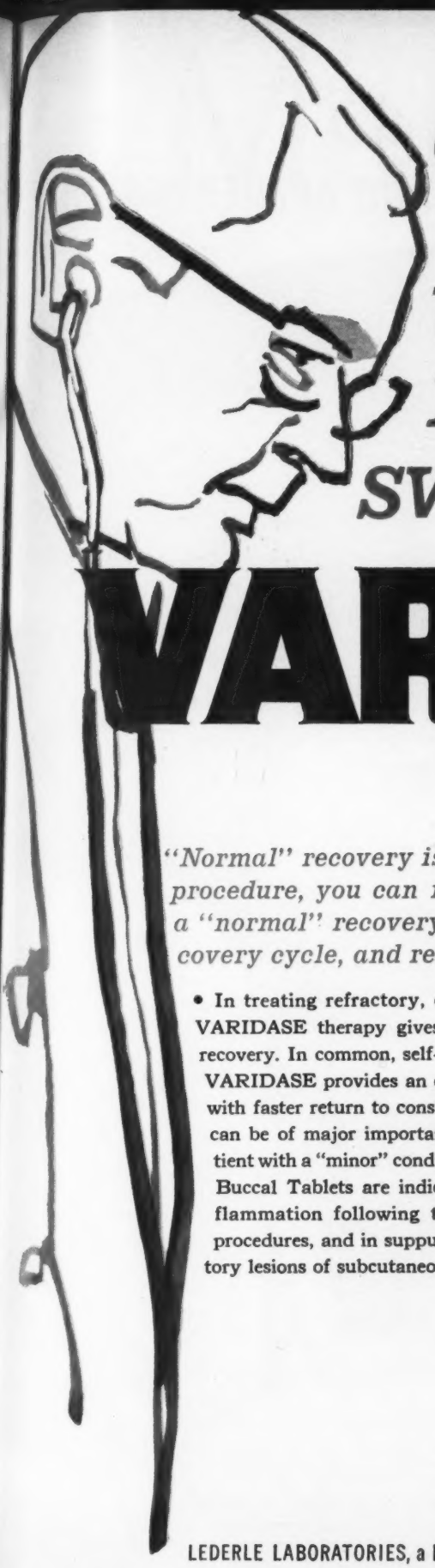
When the parents said that they have been trying very hard with Larry, recently by "pressing" him in regard to his school work, his music, going out to play with the other children, the social worker asked them to consider how Larry must feel in reaction to these efforts on the part of the parents. They seemed to realize then that not being able to come up to their expectations may make the boy feel that they are disappointed in him and do not fully love him.

As Mr. and Mrs. S talked with Larry's sister, Nancy, their eyes glowed with pride. When the social worker pointed to the difference he observed in their feelings toward the two children, they recognized, that Nancy had "the inside track," but said they could not help feeling that way under the circumstances. The worker pointed out that this was only human.

Suggesting that because Larry is a worrisome problem to them, they perhaps tended to look only at the dark side of things in regard to him, the social worker asked whether the boy did not have some good qualities. Spontaneously and positively they talked about his considerateness, his musical talent, his intelligence, his likeability. The social worker emphasized that they must have a lot of good stuff in them as parents for Larry to have these good qualities.

In the course of this interview the parents began to think together about what they could be doing to improve their relationship with Larry. When the father told about a recent incident when Larry responded well to a suggestion that he help paint his room, the worker suggested that father and mother continue such efforts to get through to Larry by doing things with him and talking with him.

Before the interview terminated a plan was made for Larry to be brought to the clinic for psychiatric evaluation in a few months and for the worker to meet again with the parents at that time. However, as the time for the evaluation approached the parents canceled the plans for this and any other further interviews. In an extensive telephone conversation Larry's mother reported that the boy's symptoms had greatly diminished, and credited this to application of the understanding she and her husband had obtained through the consultation they had received. Followup through periodic telephone conversations with Mr. and Mrs. S over the next 5 years indicated that the problem with the child had been completely resolved by the end of 6 months, and that this resolution was sustained.



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• Supplied: *Each tablet contains 10,000 Units Streptokinase, 2,500 Units Streptodornase. Boxes of 24 and 100 Tablets.*

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Mellaril is indicated for varying degrees of agitation, apprehension and anxiety in both ambulatory and hospitalized patients.

Usual starting dose: Non-psychotic patients – 10 or 25 mg. t.i.d.; Psychotic patients – 100 mg. t.i.d. Dosage must be individually adjusted until optimal response. Maximum recommended dosage: 800 mg. daily. Supply: Mellaril Tablets, 10 mg., 25 mg., 50 mg., 100 mg.

1. Sandison, R. A., Whitelaw, E., and Currie, J. D. C.: Clinical trials with Mellaril in the treatment of schizophrenia, Journal of Mental Science (British Journal of Psychiatry) 106:732, April, 1960. 2. Freed, S. C.: Thioridazine, a neuroleptic in general practice, International Record of Medicine, 172:644, Oct. 1959.

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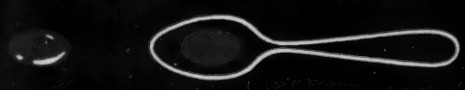

The suspension contains pyrvinium pamoate equivalent to 10 mg. pyrvinium base per cc. The sugar-coated tablets each contain pyrvinium pamoate equivalent to 50 mg. pyrvinium base. *Dosage:* Children and adults, a single oral dose equivalent to 5 mg. per Kg. of body weight. *Precautions:* Infrequent nausea and vomiting and intestinal complaints have been reported. Tablets should be swallowed whole to avoid staining teeth. Will color stools a bright red. Suspension will stain most materials.

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In this case illustration, several underlying parental problems were dealt with directly and dynamically: the distance and estrangement between father and son; the confusion and subconscious conflict between the parents which arose during the child's oedipal stage of development; the parents' resultant accumulated frustration and resentment; Larry's inability to satisfy the parents' high expectations; the parents' negative feelings toward Larry caused by his annoying symptoms and their more positive feelings toward their daughter.

In the other successful cases, similar parental problems and others were also effectively dealt with through direct and dynamic discussion.

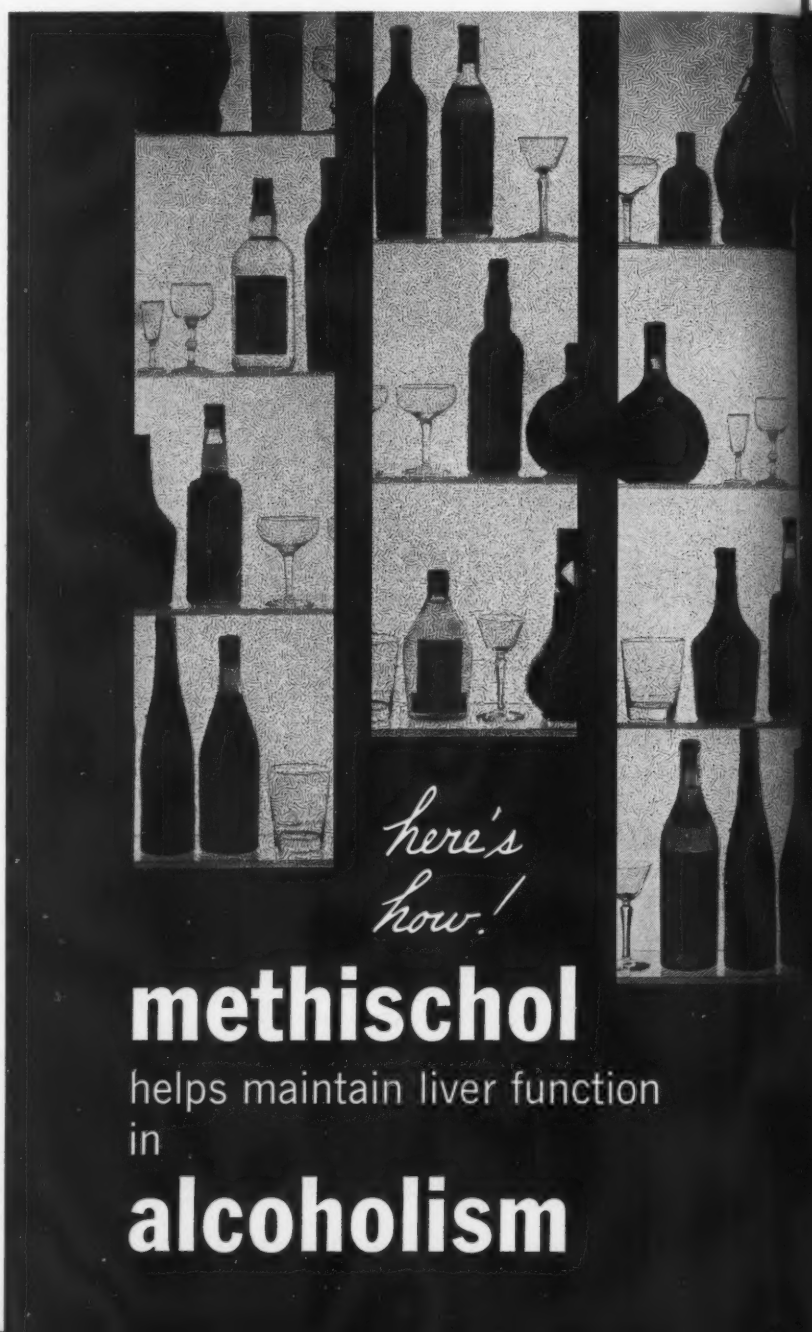
An impairment in the father-son relationship was a major problem in eight of these cases. A factor frequently encountered, as in the S case, was the father's feeling of being rejected by his child and reacting with counterrejection and discouragement. One father was deeply disappointed in his son's lack of athletic physical makeup, inclination, and ability. Another father, according to his wife, acted toward his son as a supervisor acts in a business firm. Another father became aware that he was very authoritative and strict with his son because that is how he himself was reared.

Parents' weakness in discipline was a frequently occurring problem in the successful cases. The reasons for this varied from case to case, and getting at them was an important part of the interview. A father who felt that his wife was too strict with their child was excessively lenient and permissive to counterbalance this, and the ill feeling that grew up between the parents over this difference was transferred to the child. Another couple had been unable to have a child for 8 years and consequently after a daughter was finally born to them had become extremely anxious and overprotective toward her. Consequently, the little girl developed such a demanding and controlling pattern of behavior that by the age of 4 she was the complete ruler of the household. A similar situation developed in a family with an adopted child. In another family overprotectiveness had developed after the child had had a serious accident and almost lost his life.

Another father and mother were concerned about the effeminate tendencies in their 9-year-old son, manifested by the boy's associating only with girls. The mother and the worker together helped the father bring out his worry over the possibility that perhaps he himself had an effeminate streak and that his son was taking after him. As he did so his wife helped him to see clearly the unrealities in his perception of himself. In another case a widowed mother feared that too close a relationship with her son would result in excessive feminine identification and consequent damage to his psychosexual development, and as a result she went to the other extreme of not showing him any affection. A divorced mother came to the sudden realization in the interview that she had feelings of hatred toward her son because he looked so much like his father.

In the cases that were unsuccessful, there was similar dynamic content in the initial interviews, which misled the worker into thinking that positive results could be achieved through the parent consultation approach. He was disappointed to find as time went on either that temporary or sporadic specific improvement was followed by complete return of the presenting problems; or that no improvement whatever occurred. The basic trouble lay in the incorrect evaluation of the parents and child.

For example, in one of the cases of failure, the ups and downs with respect to the child's presenting problems continued for about one year—during which there were intermittent sessions with the parents—until the social worker finally realized that there was a deep and chronic marital conflict and that the mother herself was sufficiently disturbed to require intensive psychiatric treatment. A review of the record indicated rather clearly that the worker should have been able to arrive at more accurate presumptive diagnoses of the family and of the mother early in the contact and that a



treatment plan for child and parents, with special attention to the mother's psychopathology, should have been instituted. Such cases have taught us that when we do not see progress within a few months to look for deeper disturbance in the marital relationship, the individual parent, or the child.

PSYCHIATRIC EVALUATIONS

Our failure cases point to the paramount importance of instituting effective safeguards in undertaking a program of parent consultation of this type. Psychiatric evaluation of the child as an integral part of the parent consultation process establishes the nature and degree of the child's disturbance. A finding that the disturbance is of a reactive nature adds substantially to the social worker's feeling of security in pursuing the parent consultation approach; and it is also reassuring to the parents.

The fact that the social worker sees the parents together during the child's two appointments for psychiatric evaluation provides him with an opportunity to extend and deepen his diagnostic understanding of them as individuals and of their relationship with each other. It also gives him additional opportunity to test out the parents' ability to act on the understandings they initially gained.

The process is carried forward still another step when the psychiatrist and social worker meet together with both parents in the summation interview. This direct contact of psychiatrist with the parents provides a check on the social worker's initial evaluation of the parents. Even though half the parents in the parent consultation group do not regard the psychiatric evaluation as necessary, the availability of the evaluation is a safeguard, and making use of it is strongly recommended to parents who falter in making progress with their child. Conferences between the worker and the psychiatrist who evaluates the child, supervision, and consultation are also vital safeguards.

Additional interviews beyond the summation interviews are scheduled when necessary. When the parents feel they can continue to make progress without further help, the social worker takes responsibility for keeping in touch with their progress regularly, mostly through telephone interviews.

There are, of course, weaknesses in this followup plan: a parent may try to cover up adverse developments as a self-protective measure or out of a need to please the worker; he might at the time of the telephone call be in a generally bad mood or be momentarily upset by happenings with the youngster, and so make statements that are more negative than the situation warrants; a telephone interview does not provide the interviewer with an opportunity to see the expressions and gestures that give clues to parental feelings. We believe, however, that much can be learned in the telephone interview when there has been a prior working relationship which immediately encourages openness and frankness on the telephone. The workers does not close the conversation if the parent says "everything is fine," but checks into specific aspects of the child's and the parents' interrelated problems.

The telephone interview calls for open-mindedness and objectivity on the part of the social worker which in turn intangibly encourages open-mindedness and objectivity in the parent. If there is an attitude of wishful thinking on the part of the worker this may result in the parent telling him only that which he would like to hear. Because we realize that at best the telephone interviews are only good indicators, parents are invited to come in for regular interviews when the worker has the feeling that this should be done.

The parent consultation process requires a social worker who has had enough experience with parents and children to be able to make presumptive diagnoses of the child and family; has been convinced by experience that certain kinds of parents can be reached relatively quickly and profoundly; who is creative, flexible, and able to think rapidly; and who has a considerable amount of professional security and maturity. Yet the safeguards of supervision, staff consul-

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in rheumatoid arthritis

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STEROIDS: FAR FROM ROUTINE THERAPY IN RHEUMATOID ARTHRITIS. "... it would now appear that the steroids should be employed infrequently in rheumatoid arthritis, and, when used, long-continued therapy should be avoided and the dosage reduced to the lowest possible level." [New and Nonofficial Drugs 1961, Philadelphia, J. B. Lippincott Co., 1961, p. 598.]

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... affords lasting benefits
... spares steroids
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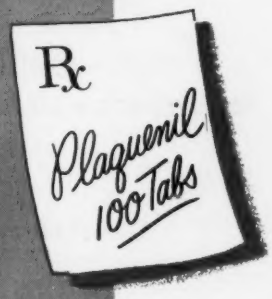
... reduces need for analgesics
... works in conjunction with both steroids and aspirin

PLAQUENIL ... produces major improvement in 60 to 83 per cent of patients
... results in a low incidence of major relapse



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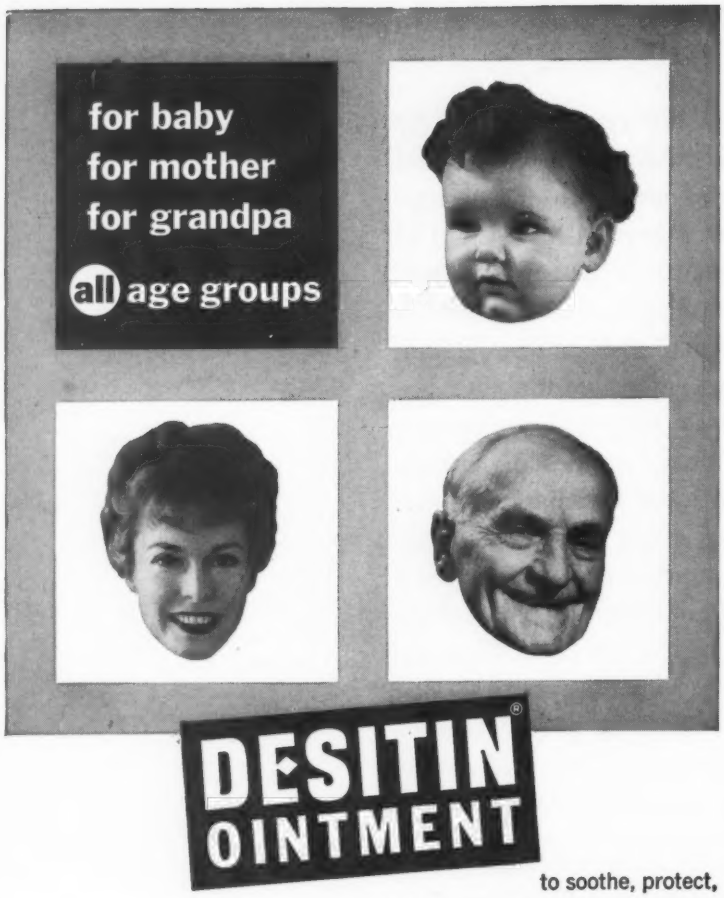

tation, and psychiatric evaluation make it possible for less experienced staff members to learn this complex and difficult process.

We believe that with the effective application of the criteria enumerated in this article and the effective implementation of the safeguards mentioned, parent consultation has a place as one of the several closely connected and interrelated approaches to family treatment.

1. McLure, Dorothy; Schreier, Harvey: Preventive counseling with parents of young children. Social Work, April 1956.
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3. Schwartz, Anne C.: Family casework in behalf of children. Social Casework, November 1959.
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Accident toll in 1960—92,000 lives*

Accidents caused about 92,000 deaths in the United States during 1960, approximately the same number as in the year before, but about 1,500 more than in 1958. When account is taken of the increase in population, however, it appears that some progress has been made in reducing the accident hazard. Provisional figures indicate that the accident death rate in the year just ended dipped slightly below the all-time low of 52.2 per 100,000 population established in 1959.

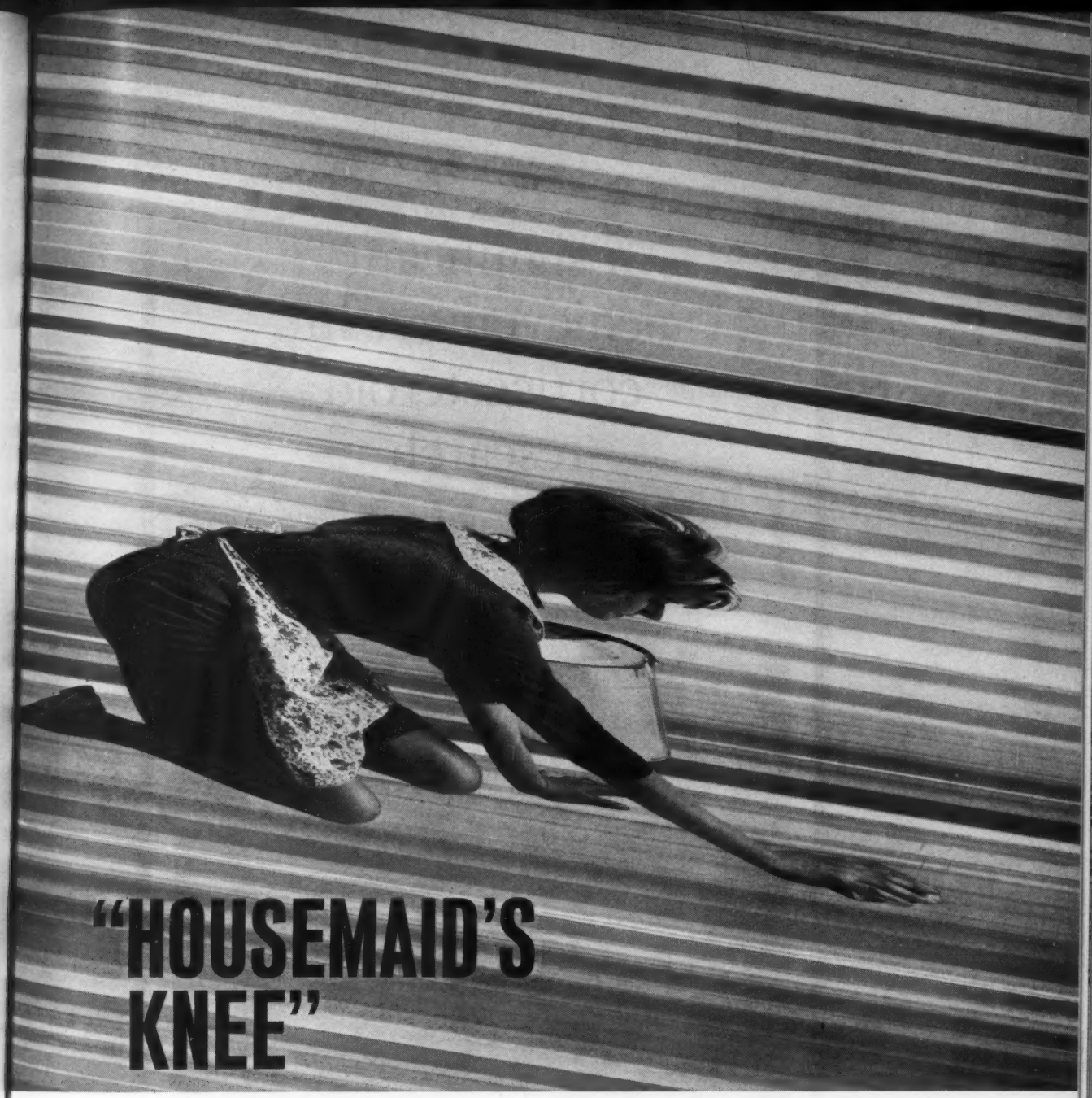
Motor vehicle accidents were responsible for the loss of approximately 38,000 lives during 1960, or a few hundred more than in 1959. This rise reflects the increase in the amount of travel; the motor vehicle accident death rate per 100 million miles of travel in 1960 may set an all-time low of 5.3.

Accidents in and about the home took 26,500 lives in 1960, or an increase of approximately 500. The annual toll from public accidents other than those involving motor vehicles continued at the level of about 16,500. The number fatally injured in accidents arising out of and in the course of employment in 1960 was little different from the figure for the year before, namely, 13,800; approximately 2,800 of these deaths resulted from motor vehicle accidents (which number is also included in the total of motor vehicle fatalities).

For Canada, only data on motor vehicle accidents are available at this time. Preliminary figures indicate that motor vehicle accidents took about 3,700 lives in the year just ended, or approximately the same as in 1959. This means that the motor vehicle accident mortality in Canada may have dropped below the rate of 21.1 per 100,000 population recorded in 1959.

Catastrophes—accidents in which five or more persons are killed—caused somewhat over 1,400 deaths in the continental United States in 1960, or about the same number as in 1959. In the year just ended there were eight major catastrophes—those which took the lives of at least 25 persons—accounting for approximately 450 fatalities. Five of the major disasters involved scheduled aviation. The worst disaster of the year was the mid-air collision between two scheduled planes over Staten Island, N. Y., on December 16, causing 134 deaths (including casualties on the ground)—the heaviest loss of life in aviation history. Another major catastrophe was the explosion of a scheduled plane in flight

*Reprinted from *Statistical Bulletin*, Metropolitan Life Insurance Company, December 1960.



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near Tell City, Ind., on March 17, taking 63 lives. The other three aviation disasters included the plane crash into Boston Harbor on October 4, which killed 62 persons; the plane crash into a swampy ravine near Richmond, Va., on January 18, which caused 50 fatalities; and the plane which "disintegrated" in flight near Wilmington, N. C., killing 34 persons on January 6—the earliest disaster of the year. On December 19, a fire swept an aircraft carrier in the Naval Shipyard at Brooklyn, N. Y., taking 50 lives. There were two natural disasters during the year just ended: Donna, the hurricane which hit the South and Northeast early in September, accounted for 30 fatalities; and the tornadoes which struck eastern Oklahoma and Arkansas on May 5 resulted in 29 deaths, most of them in Oklahoma.

Accidents involving motor vehicles were responsible for nearly one third of the approximately 1,400 lives lost in catastrophes during 1960. Civil aviation accounted for almost as many fatalities. Fires and explosions—most of them in the home—caused about one fifth of all fatal injuries sustained in catastrophes. Natural catastrophes, military aviation, and water transportation contributed appreciably to the total.

Civil aviation catastrophes took a much larger toll in 1960 than in the year before. Military aviation showed only a moderate rise in the number of fatalities. On the other hand, fatalities in catastrophic motor vehicle accidents and in fires and explosions were appreciably lower in 1960 than in 1959. The loss of life in water transportation and railroad accidents taking five or more lives was also reduced in the year just ended. Catastrophic accidents in mines and quarries accounted for approximately the same death toll in both years.

Longevity of American presidents*

When John F. Kennedy was sworn in as President of the United States on January 20 of this year, the American people had three former Chief Executives in their midst. It was the first time in three quarters of a century that so many ex-Presidents were alive at the time of an inauguration. Dwight D. Eisenhower had reached the biblical three score and ten when he turned the reins of national government over to a man 27 years his junior. Harry S. Truman was then 76 years of age, and Herbert C. Hoover had already celebrated his 86th birthday.

Happily, both Mr. Hoover and Mr. Truman have already lived for a longer time since their inauguration than would have been expected on the basis of mortality conditions prevailing at the time they took office. Since the Civil War there have been only two other Presidents —Grover Cleveland and William H. Taft —who outlived their expectations of life at inauguration.

The 15 deceased Presidents from Lincoln to Franklin D. Roosevelt lived a total of about 165 years after their inauguration; the combined expectation of life of these men on accession to the presidency was slightly over 290 years. These 15 men, in other words, lived about 8 years less, on the average, than expected. Three of these Presidents—Lincoln, Garfield, and McKinley—were assassinated while in office. But even if they are omitted from the record, the actual years lived by the deceased Presidents of the post-Civil War era still averaged 6.6 years less than expected at their inauguration.

The earlier Presidents had a much more favorable longevity record. The 15 Presidents from Washington to Buchanan lived an average of 1½ years longer than their expectation of life at inauguration. Further analysis indicates that this reflects the longevity experience of the first 10 Presidents, who outlived their expectation of life at the time they took office by an average of about 4½ years. Of the first 10, only two—Washington and William H. Harrison—fell appreciably short of their expectation of life. The expected

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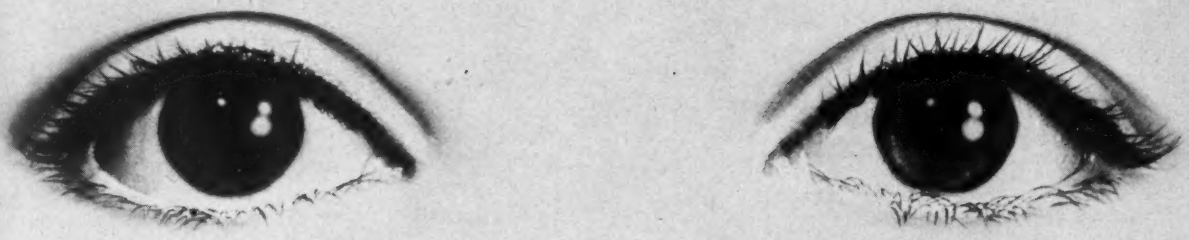

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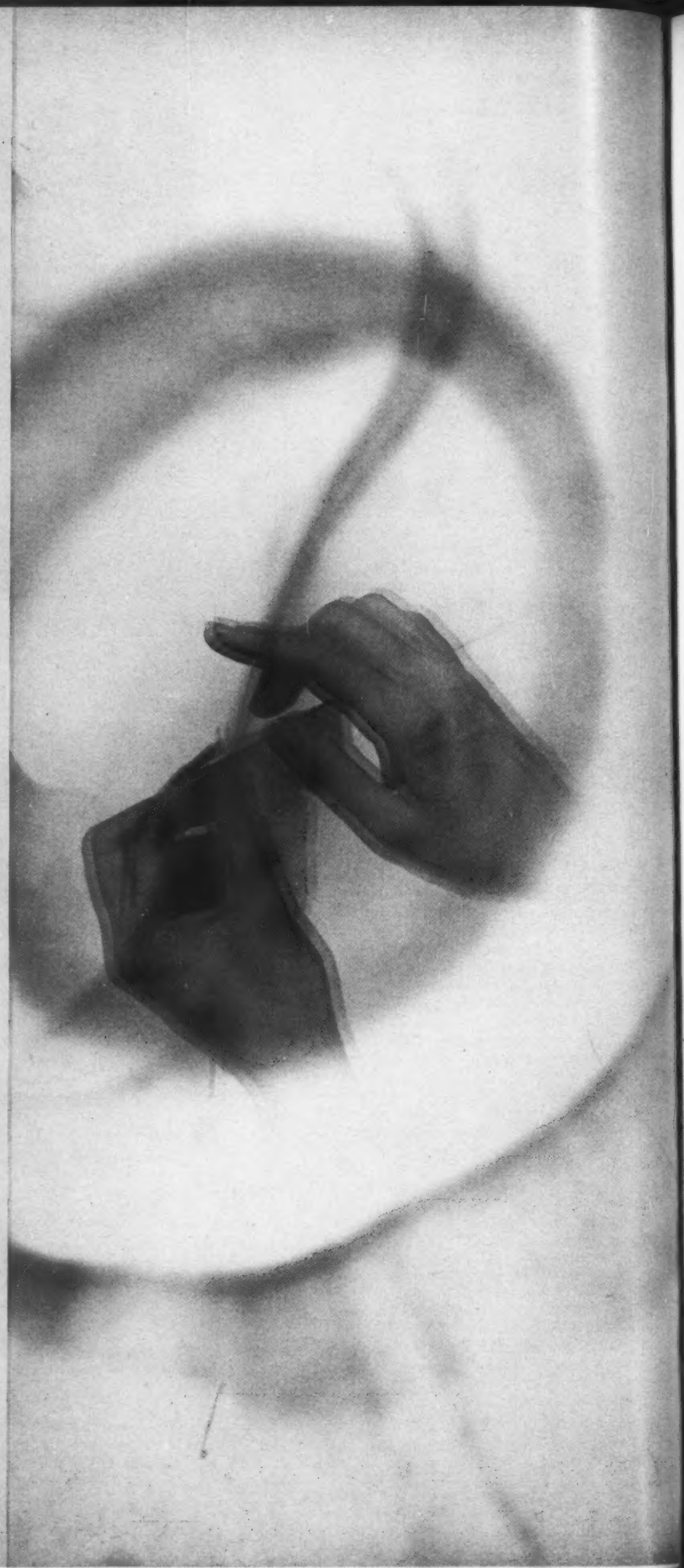

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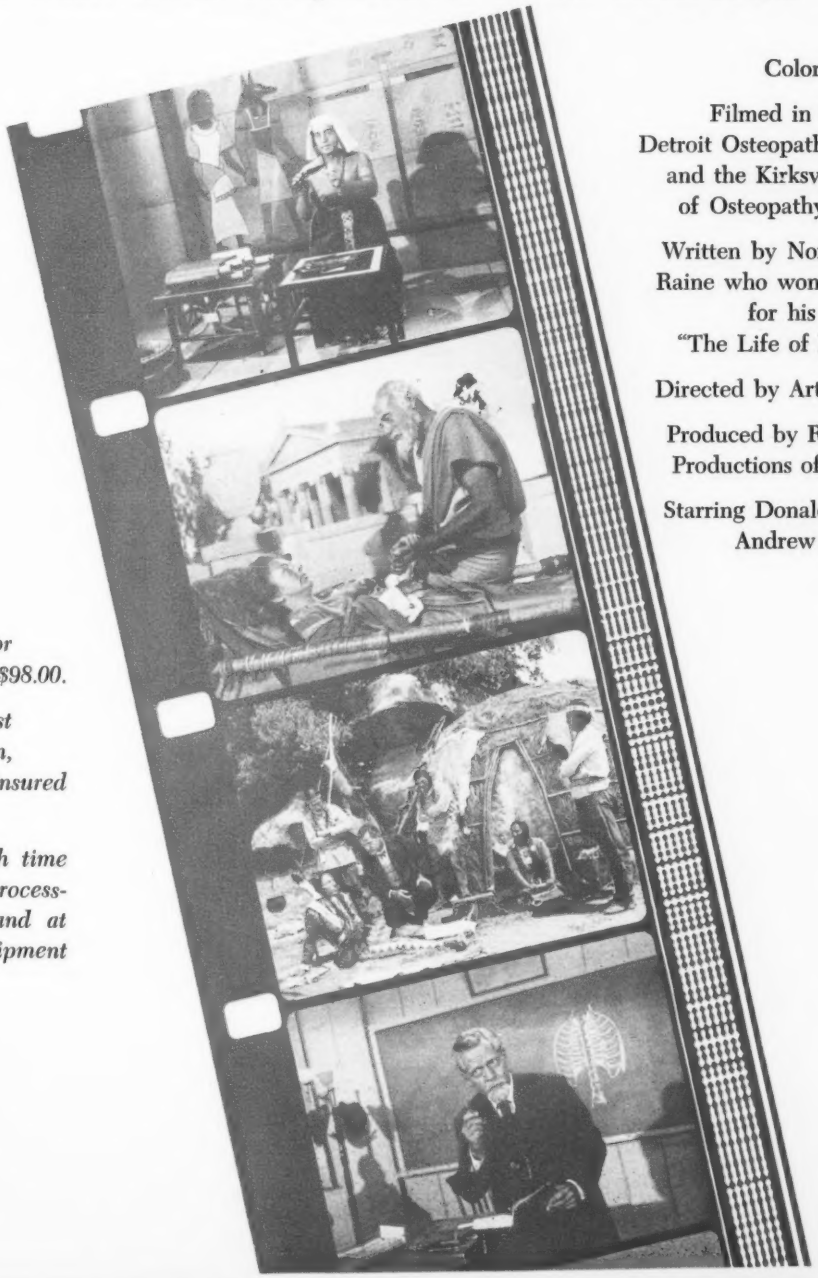

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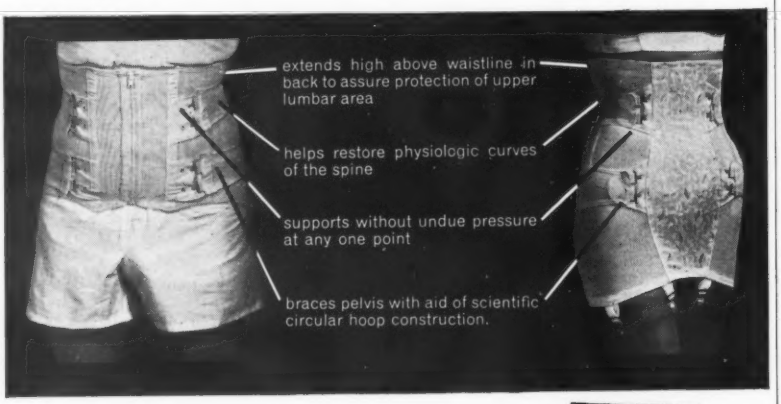
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age at death for the Presidents from Washington to Tyler averaged 72.8 years, whereas their actual age at death averaged 77.4 years.

John Adams was the longest-lived of our Presidents, living to age 90, or 16 years in excess of his expectation of life at inauguration. In a historical coincidence, Jefferson died on the same day as John Adams—July 4, 1826—at the age of 83, or 11 years later than might have been expected on the basis of the mortality prevailing at the time. Madison, like Jefferson, was 57 years of age when he took the oath of office, and lived to age 85. At the other extreme, Garfield was the shortest-lived, dying from an assassin's bullet at the age of 49; this was 19 years short of his expectation of life at inauguration. Polk and Harding died at 53 and 57, respectively, 16 years before their time. Franklin D. Roosevelt was 63 years of age when he died, 8 years short of his expectation of life at the time he took office.

Considering that there have been only 34 Presidents of the United States and that their longevity record has varied from one historic period to another, it is not possible to say whether or not the presidency shortens life.

The Constitution of the United States specifies that a person to be eligible for the presidency must have reached his 35th birthday. Actually, the youngest—Theodore Roosevelt—was 42 years old when he took office; Kennedy was 43 years of age. There were only five other Presidents in our history who were under age 50 when sworn into office: Polk, Pierce, Grant, Garfield, and Cleveland. Of the seven Presidents who had passed their 60th birthday at inauguration, five preceded Lincoln; the other two were Truman, who was 60 when he took the oath of office, and Eisenhower, who was 62. The oldest men to become President were William H. Harrison and Buchanan, who were 68 and 65 years, respectively, at their inauguration.

Incidence of disability in 1960*

During 1960, nearly one sixth of the Metropolitan's office and field personnel covered by the Company's Group insurance program was disabled by an illness or injury lasting eight days or more. The disability rate was 156 per 1,000 personnel, which was practically the same as that in the preceding two years. The stability of the experience for the group as a whole reflects contrasting trends in the disability rate for the two sexes. Among males in the age range 17-64 years, the rate decreased steadily from 114 per 1,000 in 1958 to 107 in 1960; on the other hand, among females at ages 17-59 years,† it rose from 228 to 237 per 1,000.

The relative incidence of the diseases of the respiratory system as a cause of disability lasting eight days or more decreased in both sexes during the past two years. Among females 17-59 years of age, the disability rate from these conditions fell from 102 per 1,000 in 1958 to 95 in 1960; among males aged 17-64, the rate dropped from 28 to 22 per 1,000.

Ranking second as a cause of disability among both men and women were the diseases of the digestive system. Although the disability rate for each sex was greater at ages 17-24 than in the next older age group, the highest rate was recorded at ages 45 and over.

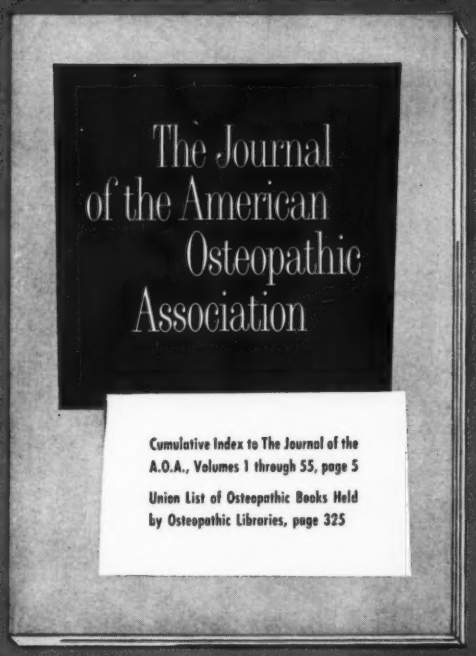
The diseases of the circulatory system rank third as a cause of disability lasting eight days or more among men, but among women they are only a relatively minor cause of disabling illness.

Disabling accidents are considerably more common among women than among men at age 25 and over.

The other leading causes of disability —diseases of the genito-urinary system, diseases of the bones and other organs of movement, neoplasms, and psychoneuroses and psychoses—likewise are much more frequent among women than among men.

*Reprinted in part from *Statistical Bulletin*, Metropolitan Life Insurance Company, March 1961.

†Normal retirement age for women is five years below that for men.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Cumulative Index to The Journal of the A.O.A., Volumes 1 through 55, page 5
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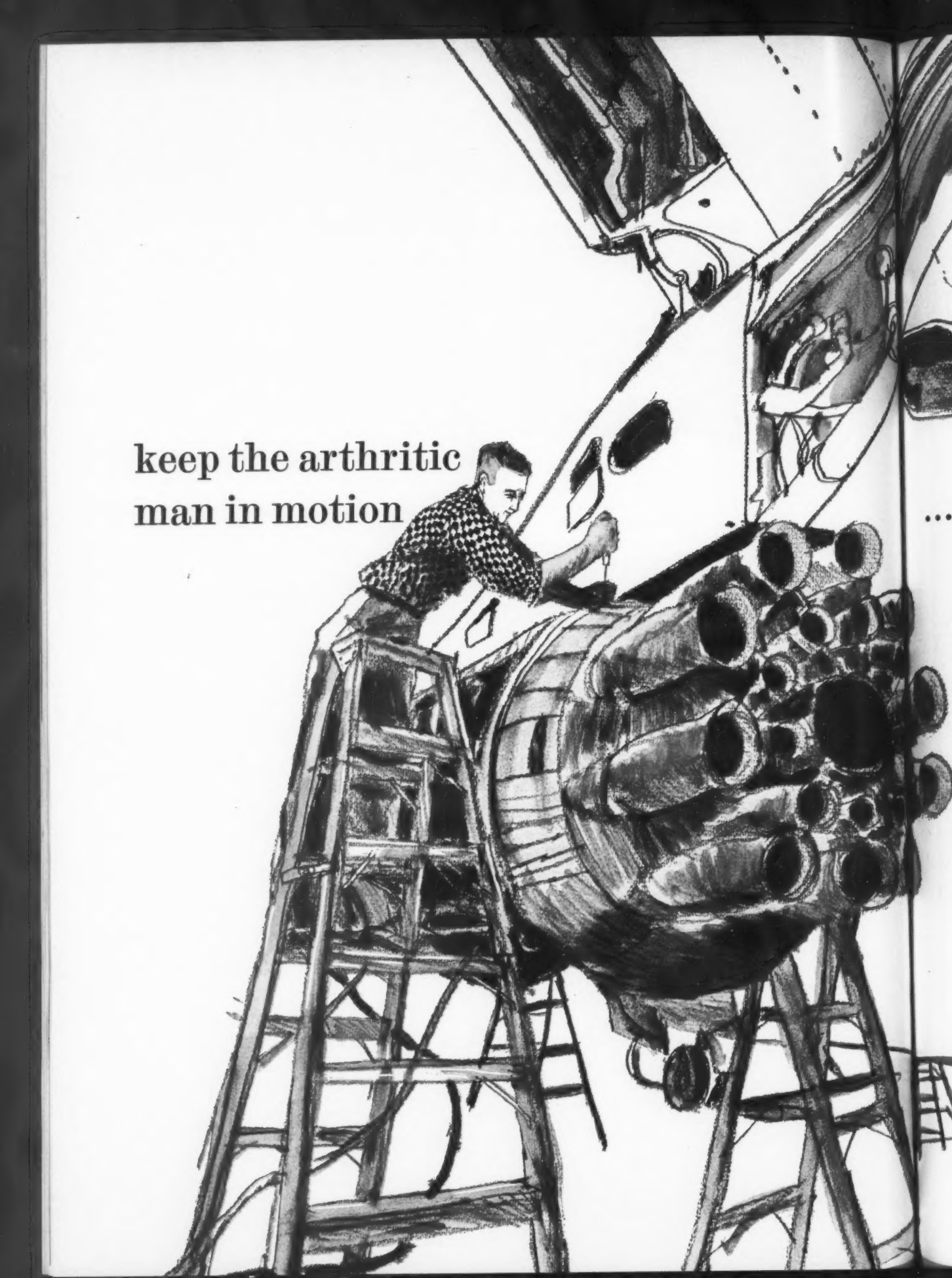
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1. Ernst, E. M.: Pennsylvania M. J. *63*:708 (May) 1960. 2. Settel, E.: Clin. Med. *7*:1835 (Sept.) 1960.

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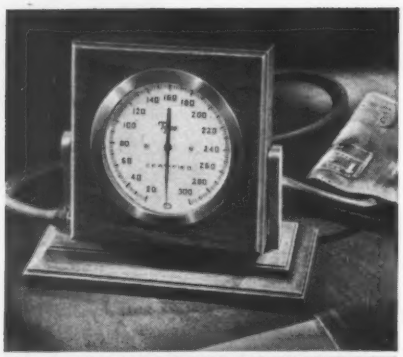

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Books received

Books received for review during the period from May 5 to June 5 are listed below. Reviews will be published as space permits.

DISEASES OF THE INTERVERTEBRAL DISC AND ITS SURROUNDING TISSUES. By Reuben Rabinovitch, B.A., M.Sc., M.D., Assistant Professor in Neurology and Neurosurgery, McGill University, Faculty of Medicine, Montreal; Assistant Neurologist, The Department of Neurology and Neurosurgery, The Royal Victoria Hospital and The Montreal Neurological Institute. Cloth. Pp. 152, with illustrations. Price $8.50. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Illinois, 1961.

MAN'S CONCERN FOR HIS FELLOW MAN. By Arnaud C. Marts. Cloth. Pp. 64, with illustrations. Price $2.00. Marts & Lundy, 521 Fifth Avenue, New York 17, 1961.

Kimber-Gray-Stackpole-Leavell's ANATOMY AND PHYSIOLOGY, with an accompanying Teacher's Guide. Edited by Lutie C. Leavell, M.A., M.S., Civilian Consultant, U.S. Army Nurse Corps, Walter Reed Institute of Research, Washington, D.C.; Lecturer, School of Nursing, University of Pennsylvania; Professor Emeritus of Nursing Education, Teachers College, Columbia University, New York. Ed. 14. Cloth. Pp. 779, with illustrations. Price $6.95. The Macmillan Company, 60 Fifth Avenue, New York 11, 1961.

THE HUMAN CEREBELLUM. An Atlas of Gross Topography in Serial Sections. By Jay B. Angevine, Jr., Ph.D., Associate in Anatomy, Harvard Medical School; Elliott L. Mancall, M.D., Assistant Professor of Neurology, Jefferson Medical College; formerly Research Fellow in Neurology, Harvard Medical School; and Paul I. Yakovlev, M.D., Clinical Professor of Neuropathology and Curator of the Warren Anatomical Museum, Harvard Medical School. Cloth. Pp. 138, with illustrations. Price $15.00. Little, Brown and Company, 34 Beacon Street, Boston 6, 1961.

NEURORADIOLOGY WORKSHOP. Vol. I. Scalp, Skull and Meninges. By Leo M. Davidoff, M.D., Active Consultant Neurosurgeon, Montefiore Hospital; Professor and Chairman, Department of Neurosurgery, Albert Einstein College of Medicine, Yeshiva University, New York; Harold G. Jacobson, M.D., Chief, Division of Diagnostic Radiology, Montefiore Hospital; Professor of Clinical Radiology, New York University School of Medicine, New York; and Harry M. Zimmerman, M.D., Chief, Division of Laboratories, Montefiore Hospital; Professor of Pathology, College of Physicians and Surgeons, Columbia University, New York. Cloth. Pp. 256, with illustrations. Price $16.50. Grune & Stratton, Inc., 381 Park Avenue South, New York 16, 1961.

RECENT ADVANCES IN HUMAN NUTRITION. With special reference to Clinical Medicine. By J. F. Brock, D.M. (Oxon.), F.R.C.P. (Lond.), Professor of Medicine, University of Cape Town, Cape Town, South Africa. Cloth. Pp. 454, with illustrations. Price $11.50. Little, Brown and Company, 34 Beacon Street, Boston 6, 1961.

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*Lawson, G. W.: Postgrad. Med. 22:501 (Nov.) 1957

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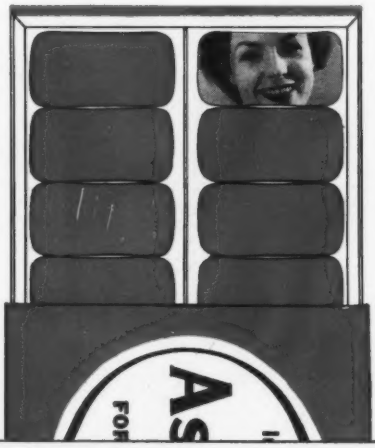

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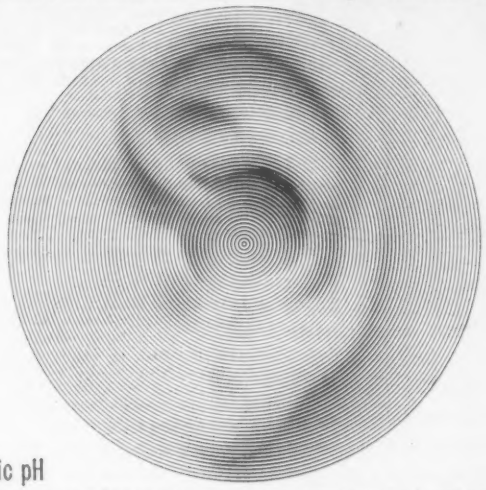

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*Granberry, C., and Beatrous, W. P.: E.E.N.T. Mo. 36:294 (May) 1957.

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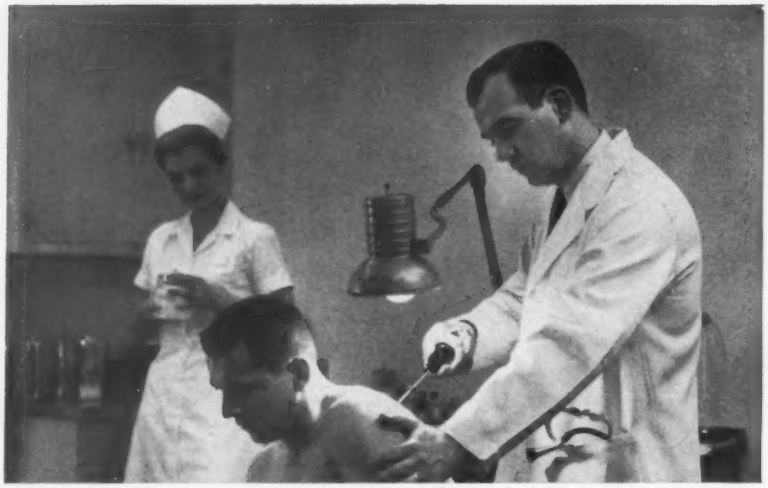

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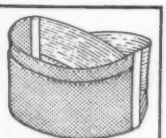

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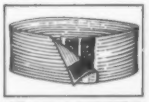

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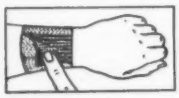

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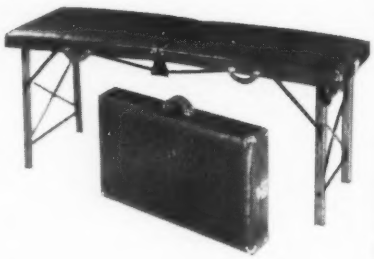

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tion, medical organizations and literature, incidence of illness and economic aspects of medical practice. Compiled by Peter S. Nagan, A.B., M.A., M.S. Paper. Pp. 528, with illustrations. Price $5.00. W. B. Saunders Company, West Washington Square, Philadelphia 5, 1961.

SURGERY OF THE ANUS, RECTUM AND COLON. By J. C. Goligher, Ch.M., F.R.C.S., Professor of Surgery, University of Leeds, and Surgeon, General Infirmary at Leeds; formerly Surgeon, St. Mary's Hospital and St. Mark's Hospital for Diseases of the Rectum and Colon, London. Cloth. Pp. 829, with illustrations. Price $35.00. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Illinois, 1961.

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SYMPTOM DIAGNOSIS. By Wallace Mason Yater, A.B., M.D., M.S. (in Med.), F.A.C.P., Director, Yater Clinic, Washington, D.C.; formerly Professor of Medicine and Director of the Department of Medicine, Georgetown University School of Medicine; Physician-in-Chief, Georgetown University Hospital; Physician-in-Chief, Gallinger Municipal Hospital, Washington, D.C.; and William Francis Oliver, B.S., M.D., F.A.C.P., Assistant Clinical Professor of Medicine, University of Southern California School of Medicine; Consultant, Santa Barbara General Hospital; Attending Physician, Cottage Hospital and St. Francis Hospital, Santa Barbara, California; Attending Physician, Service of Internal Medicine, County of Los Angeles General Hospital. Ed. 5. Cloth. Pp. 1035, with illustrations. Price $15.00. Appleton-Century-Crofts, Inc., 35 West 32nd Street, New York 1, 1961.

EXISTENTIALISM AND PSYCHIATRY. Four lectures. By Rudolf Allers, M.D., Ph.D., Professor of Philosophy (Emeritus), Georgetown University, Washington, D.C. Cloth. Pp. 103. Price $5.50. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Illinois, 1961.

MANAGEMENT OF OBSTETRIC DIFFICULTIES. Revised by J. Robert Willson, M.D., M.S., Professor of Obstetrics and Gynecology, Temple University School of Medicine; Head of the Department of Obstetrics and Gynecology, Temple University Medical Center. Ed. 6. Cloth. Pp. 687, with illustrations. Price $16.50. The C. V. Mosby Company, 3207 Washington Boulevard, St. Louis 3, 1961.

SYSTEMATIC OBSERVATION OF GROSS HUMAN BEHAVIOR. By G. R. Pascal and W. O. Jenkins, Professors of Psychology, University of Tennessee. Cloth. Pp. 126, with illustrations. Price $4.75. Grune & Stratton, Inc., 381 Park Avenue South, New York 16, 1961.

CANCER CHEMOTHERAPY. Prepared by the Staff of The University of Texas, M. D. Anderson Hospital and Tumor Institute, Texas Medical Center, Houston, Texas. Under the direction of R. Lee Clark, Jr., M.D., D.Sc. (Hon.) Cloth. Pp. 253, with illustrations. Price $10.50. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Illinois, 1961.

ESSENTIAL PATHOLOGY. By Roger D. Baker, M.D., Professor of Pathology, Duke University School of Medicine; Chief of Laboratory Service, Veterans Administration Hospital, Durham, North Carolina. Cloth. Pp. 638, with illustrations. Price $9.50. The Williams & Wilkins Company, 428 East Preston Street, Baltimore 2, 1961.

TECHNIQUES IN TROPICAL PATHOLOGY. By B. G. Maegraith, Professor of Tropical Medicine; W. E. Kershaw, V.R.D., Professor

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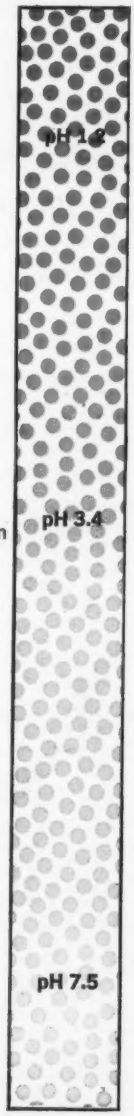

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Medrol benefits (anti-inflammatory, anti-allergic, antirheumatic, antileukemic, anti-hemolytic) have been demonstrated in acute rheumatic carditis, rheumatoid arthritis, asthma, hay fever and allergic disorders, dermatoses, blood dyscrasias, and ocular inflammatory disease involving the posterior segment.

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1. Dugger, J. A.: J. Michigan M. Soc. *59*:1812 (Dec.) 1960.

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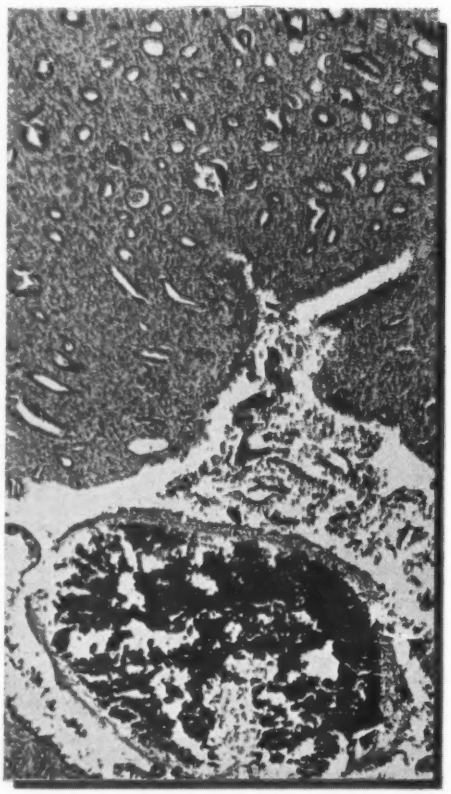
GOUT

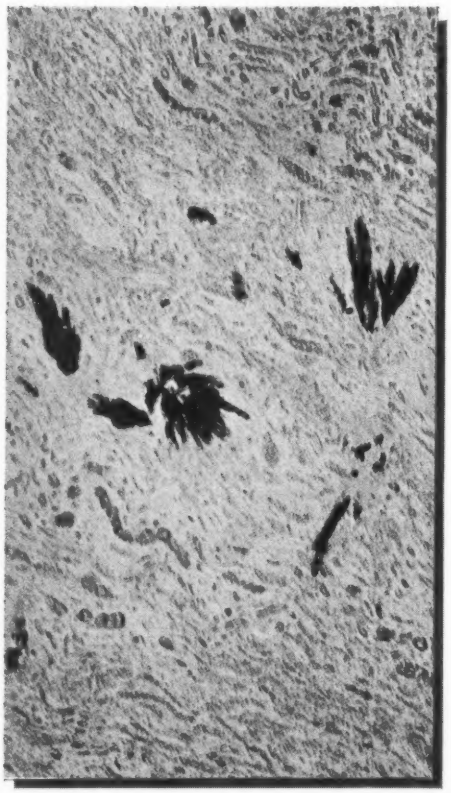
"For centuries the victims of gout have been the subject of lampoons and caricatures. We now know that they should rather be the objects of great concern, for the disease is painful, disabling and is accompanied by complications that impair health and shorten life."[1]

Kidney impairment, with varying degrees of hypertension and arteriosclerosis, is the critical complication of gout. "From 30 to 50 per cent of gouty patients are said to die of renal disease."[2]

Tophus in the calyx, surrounded by fibrinoid exudate and detached mucosal epithelium.[3]



The mid- and outer portion of the pyramid with typical uric acid crystals in the collecting system.[3]



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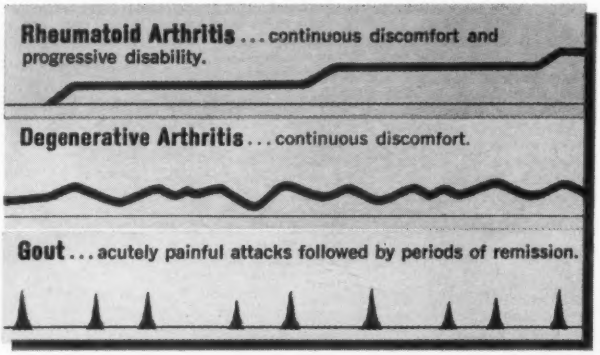
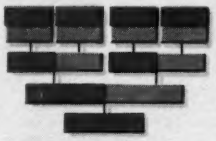

Rheumatoid Arthritis ... continuous discomfort and progressive disability.
Degenerative Arthritis ... continuous discomfort.
Gout ... acutely painful attacks followed by periods of remission.



"If a family history of gout is obtained, even though it is one or two generations removed, this information is significant."[4]

"The metatarsophalangeal joint of a great toe is affected early or repeatedly in some cases, rarely or never in others."[6]

"All patients complaining of non-traumatic musculoskeletal discomfort should have at least one serum uric acid determination."[5]

Colchicine test: "Colchicine should be administered as early as possible after the onset of articular distress, 1 mg.... every 2 hours until the onset of gastro-intestinal distress. From 5 to 8 mg. usually are required."[9] Pain relief is highly indicative of gout.

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1. Cornish, A. L.: J. Kentucky M.A. 58:707, June, 1960. 2. Wyngaarden, J. B.: Arthritis & Rheumatism 1:191, June, 1958. 3. Talbott, J. H. and Terplan, K. L.: Medicine 39:405, Dec., 1960. 4. Talbott, J. H.: Gout, New York, Grune & Stratton, 1957, p. 123. 5. Kuzell, W. C., et al.: J. Chron. Dis. 2:645, Nov., 1955. 6. Hench, P. S.: Gout and gouty arthritis, in Cecil, R. L.: A textbook of medicine, ed. 10, Phila., W. B. Saunders Co., 1959. 7. Bartels, E. C., and Kepkay, P. H.: Bull. Vancouver M.A. 29:306, April, 1953. 8. Boland, E. W.: World-Wide Abstracts of Gen. Med. 3:16, Jan., 1960. 9. Talbott, J. H.: Current Med. Dig. 26:57, Nov., 1959.

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of Parasitology and Entomology; and D. Dagnall, Chief Technician and Demonstrator in Parasitology from the Liverpool school of Tropical Medicine. Cloth. Pp. 164, with illustrations. Price $6.00. Oliver and Boyd, London. Charles C Thomas, Publisher, exclusive U.S. distributors, 301-327 East Lawrence Avenue, Springfield, Illinois, 1961.

HEMATOLOGY IN PRACTICE. By Steven O. Schwartz, M.S., M.D., Professor of Medicine, Northwestern University Medical School; Professor of Medicine, Cook County Graduate School of Medicine; Attending Hematologist, Cook County Hospital and Director Department of Hematology, Hektoen Institute for Medical Research of the Cook County Hospital; Senior Attending Physician, Chicago Wesley Memorial Hospital; Wilson H. Hartz, Jr., M.D., Instructor in Medicine, Northwestern University Medical School; Associate Attending Physician, Department of Medicine, Cook County Hospital; Associate Attending Physician, Department of Medicine, Chicago Wesley Memorial Hospital; and Joseph H. Robbins, M.D., Instructor in Medicine, Northwestern University Medical School; Associate Attending Physician, Department of Medicine, Cook County Hospital. Cloth. Pp. 329, with illustrations. Price $14.00. McGraw-Hill Book Company, Inc., 330 West 42nd Street, New York 36, 1961.

ILLUSTRATIONS OF THE NERVOUS SYSTEM. Atlas III. By Louis Hausman, M.D., Professor of Clinical Medicine (Neurology), Emeritus. Cornell University Medical College; Clinical Professor of Neuropsychiatry, New York University College of Medicine; formerly Director of the Neurological Service, Second (Cornell) Medical Division, Bellevue Hospital. Cloth. Pp. 168, with illustrations. Price $9.50. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Illinois, 1961.

HEALTH ORGANIZATIONS OF THE UNITED STATES AND CANADA: NATIONAL REGIONAL AND STATE. A Directory of Voluntary Associates, Professional Societies and other Groups Concerned with Health and Related Fields. By Clara Sedacca Wasserman with Paul Wasserman. Cloth. Pp. 191. Price $10.00. Publications Section, Graduate School of Business and Public Administration, Cornell University, Ithaca, New York, 1961.

HYPOKINETIC DISEASE. Diseases Produced by Lack of Exercise. By Hans Kraus, M.D., Associate Professor, Physical Medicine and Rehabilitation, New York University; and Wilhelm Raab, M.D., F.A.C.P., F.A.C.C., F.C.C.P., F.A.C.S.M., Active Emeritus Professor of Experimental Medicine and Director of the Cardiovascular Research Unit, University of Vermont College of Medicine; Attending Physician, DeGoesbriand Memorial Hospital, Burlington, Vermont. Cloth. Pp. 193, with illustrations. Price $7.50. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Illinois, 1961.

RECOGNIZING THE DEPRESSED PATIENT. With Essentials of Management and treatment. By Frank J. Ayd, Jr., M.D., Diplomate, American Board of Neurology and Psychiatry; Fellow, American Psychiatric Association; Chief of Psychiatry, Franklin Square Hospital, Baltimore, Maryland. Cloth. Pp. 138, with illustrations. Price $3.75. Grune & Stratton, Inc., 381 Park Avenue South, New York 16, 1961.

CHEMISTRY AND THERAPY OF CHRONIC CARDIOVASCULAR DISEASE. By Richard J. Jones, A.B., M.A., M.D., Associate Professor, Department of Medicine, University of Chicago; and Louis Cohen, B.S., M.D., Instructor, Department of Medicine, University of Chicago; Advanced Research Fellow, American Heart Association. Cloth. Pp. 200, with illustrations. Price $7.50. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Illinois, 1961.

INFLAMMATION AND DISEASES OF CONNECTIVE TISSUE. A Hahnemann Symposium. Edited by Lewis C. Mills, M.D., Head, Section of Endocrinology and Metabolism, Hahnemann Medical College and Hospital; and John H. Moyer, M.D., Professor and Chairman of the Department of Medicine, Hahnemann Medical College and Hospital. Cloth. Pp. 900, with illustrations. Price 16.00. W. B. Saunders Company, West Washington Square, Philadelphia 5, 1961.

CEREBRAL ANOXIA AND THE ELECTROENCEPHALOGRAM. The Proceedings of the Marseille Colloquium, sponsored by the Reunion Europeéne d'Information Eleectróencéphalographique, and organized by Professor H. Gastaut and Dr. H. Fischgold. Edited by Henri Gastaut, M.D., Professor and Director, Laboratories of Neurobiology, Faculté de Médecine, Institut National d'Hygiène, Marseille, France; and John Stirling Meyer, B.S., M.Sc., M.D., C.M., Professor and Chairman, Department of Neurology, Wayne State University, College of Medicine, Detroit, Michigan. Cloth. Pp. 617, with illustrations. Price $24.50. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Illinois, 1961.

PROGRESS IN CLINICAL SURGERY. Edited by Rodney Smith, M.S., F.R.C.S., Surgeon, St. George's Hospital, London. Series II. Cloth. Pp. 327, with illustrations. Price $11.50. Little, Brown & Company, 34 Beacon Street, Boston 6, 1961.

RESUSCITATION OF THE UNCONSCIOUS VICTIM. A Manual for Rescue Breathing. By Peter Safar, M.D., Chief, Department of Anesthesiology, Baltimore City Hospitals; Assistant Professor of Anesthesiology, The Johns Hopkins University School of Medicine; Clinical Associate Professor of Anesthesiology, University of Maryland School of Medicine; and Martin C.

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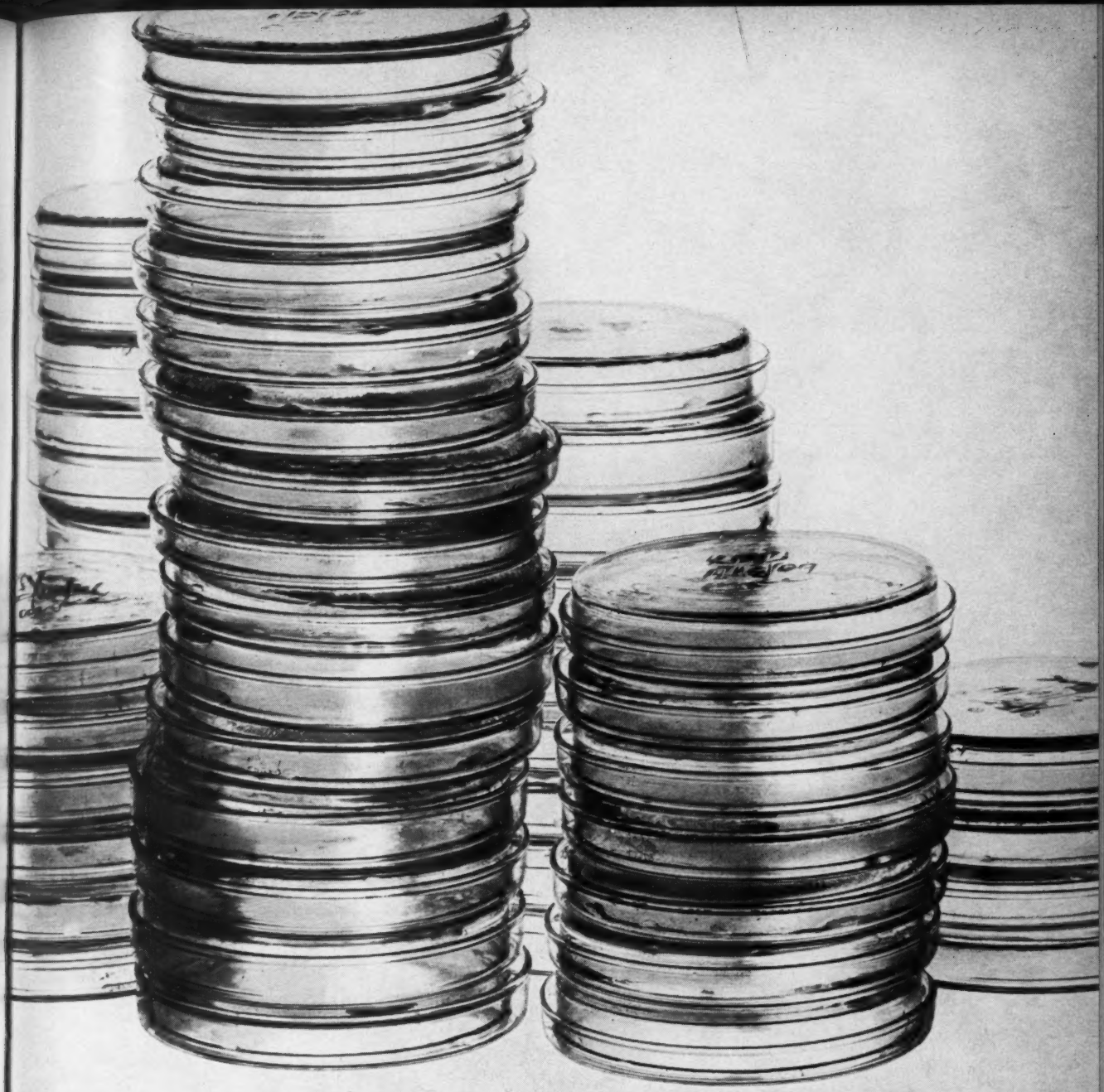

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1. Scheifley, C. H.: Proc. Staff Meet. Mayo Clin. 34:408 (Aug. 19) 1959.
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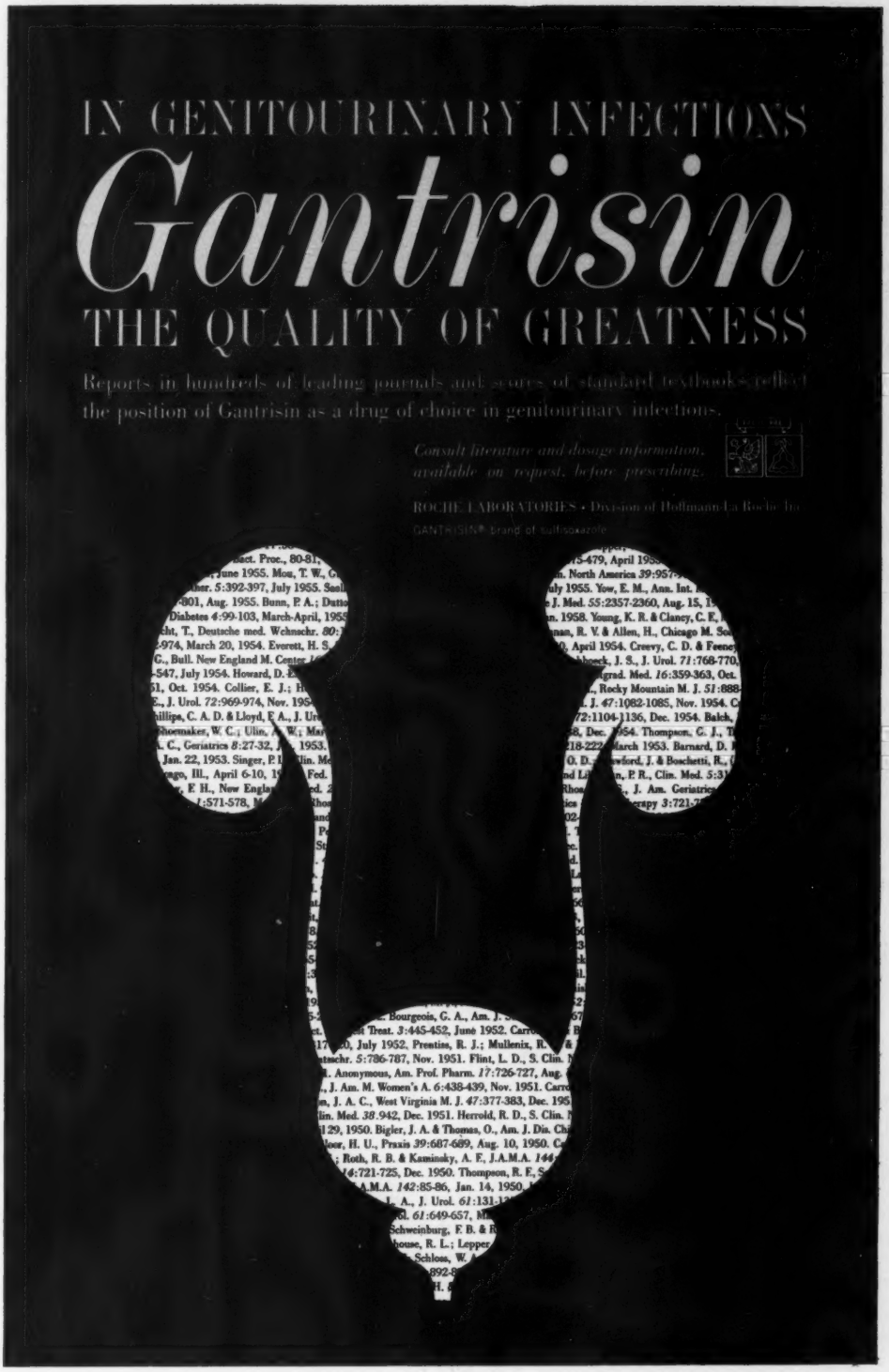

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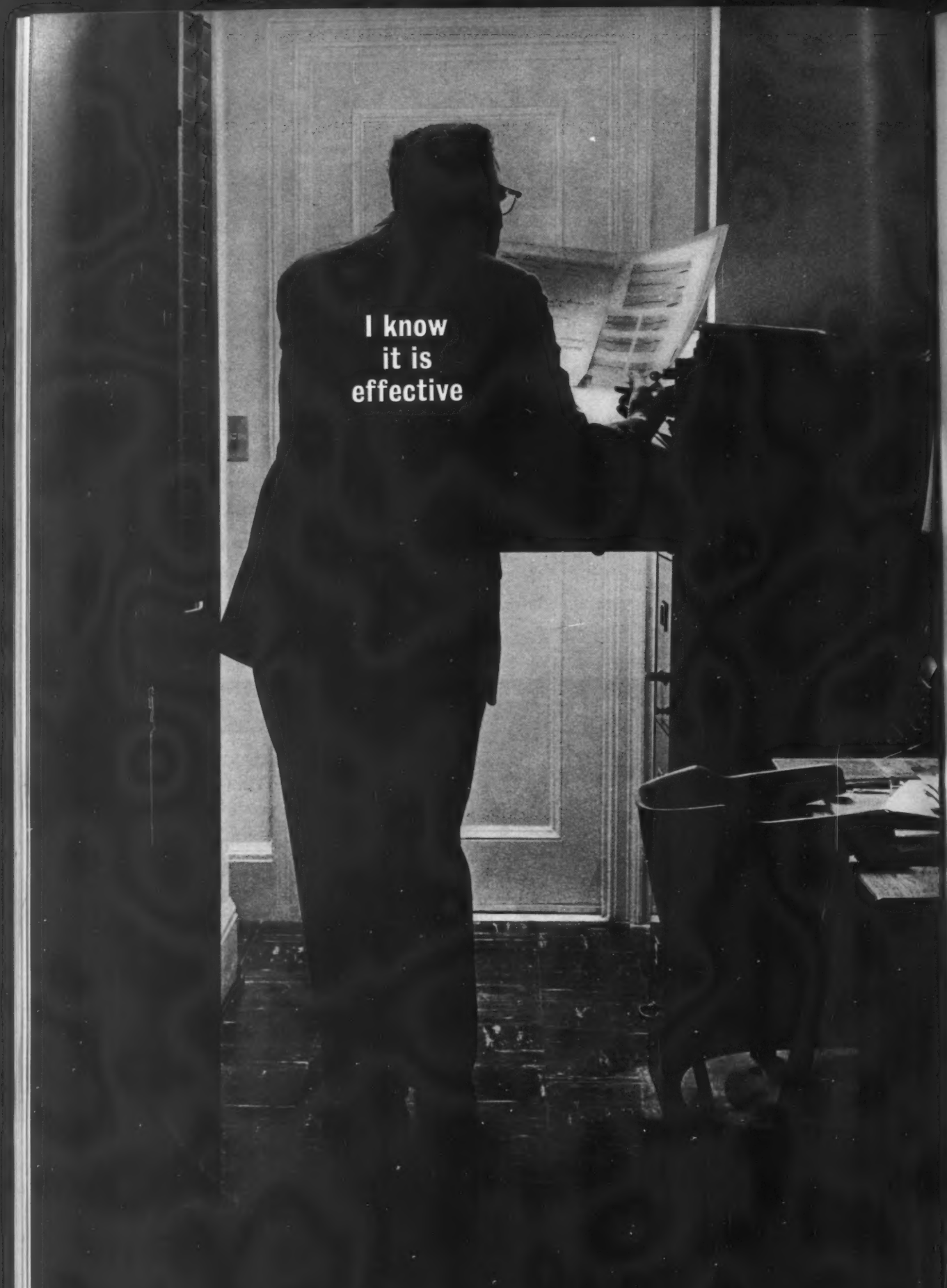
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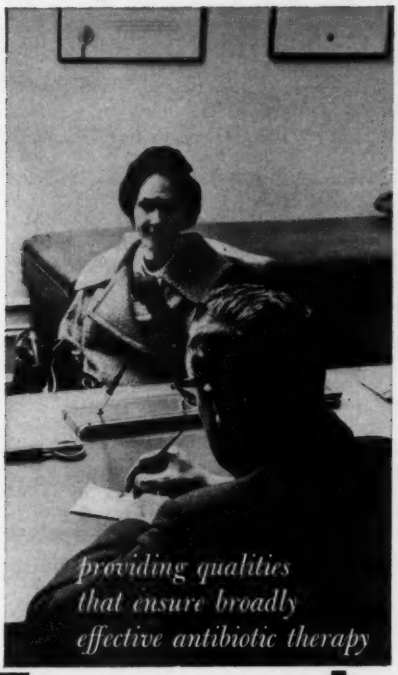

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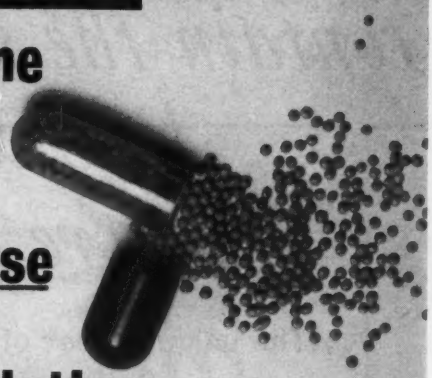

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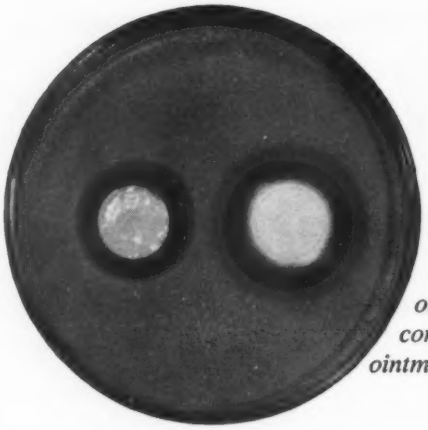

Neo-Polycin Ointment on right conventional grease-base ointment on left

Agar incubations show Neo-Polycin® Ointment more effective against common topical pathogens

This agar plate, containing *Staph. aureus*, was incubated for 24 hours at room temperature. Note the greater zone of inhibition around Neo-Polycin Ointment (right), than around the grease-base ointment of comparable antibiotic content (left). Tests on the following pathogens gave similar results: *beta hemolytic strep., E. coli, Proteus vulgaris, Pseudomonas aeruginosa.* Higher concentrations of antibiotics released from Neo-Polycin tend to inhibit the growth of relatively resistant strains and minimize antibacterial resistance created by sub-effective concentrations.

formula: Each gram of Neo-Polycin Ointment contains 4.28 mg. neomycin sulfate (3 mg. neomycin base), 400 units zinc bacitracin, 8000 units polymyxin B sulfate.

*FUZENE®—a patented base which is miscible with blood, pus, tissue exudates.

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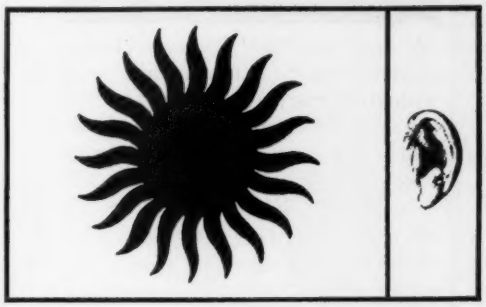

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after 3 years' clinical experience:
here is what we now know about MER/29 and . . .

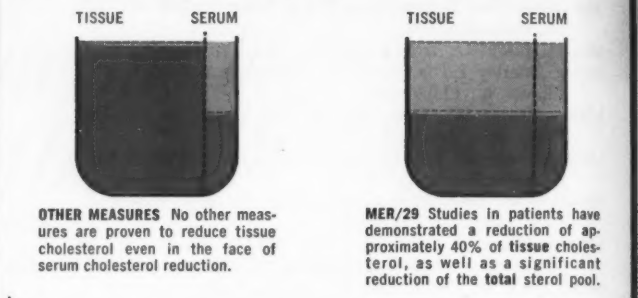
WE KNOW HOW MER/29 ACTS

We know that MER/29 lowers cholesterol in 8 out of 10 patients, even without dietary restrictions. In 576 patients studied by various physicians, average cholesterol levels dropped from 303 mg. % to 241 mg. % —an average decrease of 62 mg. %.

We know that MER/29 reduces total sterols in both blood and tissue.

We know that MER/29 does this by inhibiting the body's own production of cholesterol.

We know that its use in over 300,000 patients reaffirms the safety margins established in early laboratory and clinical data.



OTHER MEASURES No other measures are proven to reduce tissue cholesterol even in the face of serum cholesterol reduction.

MER/29 Studies in patients have demonstrated a reduction of approximately 40% of **tissue** cholesterol, as well as a significant reduction of the **total** sterol pool.

effect on
body pool of cholesterol

WE KNOW THE BENEFITS OF MER/29

We know that, in some patients, concurrent clinical benefits attend the use of MER/29. Published papers on MER/29 therapy to date report improvement in 50 of the 79 anginal patients reported in these studies, and comparable results are being obtained in similar studies now in progress. Among the other benefits reported are:

decreased incidence and severity of anginal attacks

improved* ECG *patterns

diminished nitroglycerin dependence

increased sense of well-being

"During triparanol [MER/29] therapy there was a definite improvement in the electrocardiographic tracings in response to exercise in 3 of 11 subjects with angina pectoris." —*Hollander, W.*, et al.: *J.A.M.A.* 174:*5 (Sept. 3) 1960.*

"Nitroglycerine requirements decreased in 3 [of 5 outpatient] patients, including the patient showing electrocardiographic improvement....Three [of 4 private male patients], after a lapse of some weeks, showed improvement in exercise electrocardiograms, which was sustained but not further improved in subsequent observations." —*Corcoran, A. C.*, et al.: *Progr. Cardiovasc. Dis.* 2: *(Pt.1) 576 (May) 1960.*

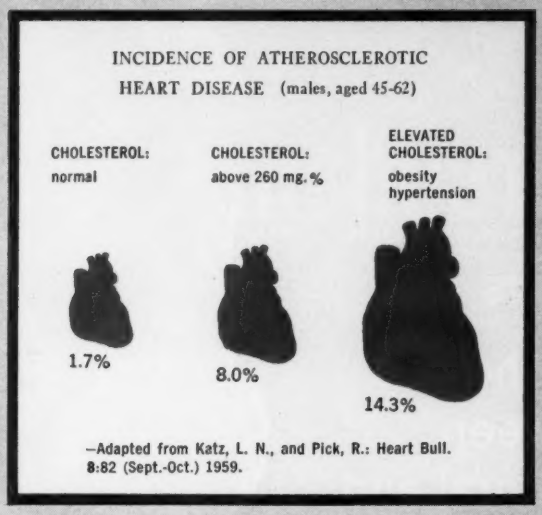
"Of the 45 patients with coronary artery disease followed for 1 year, 16 had a history of frequent anginal attacks. Fourteen of these spontaneously stated that their angina disappeared within 2 months of [MER/29] therapy....In one patient...with persistent coronary insufficiency pattern (ST segment depressions in multiple leads), there was a complete reversion to a normal tracing during MER/29 therapy with associated clinical improvement in angina." —*Lisan, P.: Progr. Cardiovasc. Dis.* 2: *(Pt. 1) 618 (May) 1960.*

....what we are learning about atherosclerosis

WHY MER/29 MAY FAVORABLY ALTER ATHEROSCLEROSIS

"It has become generally accepted that elevated blood cholesterol or lipid, if sustained long enough, leads to early atherosclerosis."
–Page, I. H.: Mod. Med. 29:71 (Mar. 20) 1961.

Epidemiologic studies show that low cholesterol levels are associated with low incidence of atherosclerosis and coronary artery disease. On the basis of such studies, Stamler has said: "... a 15 to 20 per cent reduction in mean serum cholesterol levels alone might be associated with a 25 to 50 per cent reduction in coronary disease incidence rates in middle-aged men."
–Stamler, J.: Am. J. Pub. Health 50:(Pt. 2) 14 (Mar.) 1960.


INCIDENCE OF ATHEROSCLEROTIC HEART DISEASE (males, aged 45-62)
CHOLESTEROL: normal
CHOLESTEROL: above 260 mg. %
ELEVATED CHOLESTEROL: obesity hypertension
1.7%
8.0%
14.3%
–Adapted from Katz, L. N., and Pick, R.: Heart Bull. 8:82 (Sept.-Oct.) 1959.

THE DECISION FACING THE PHYSICIAN

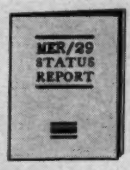
Despite our knowledge of the action, benefits and safety of MER/29, much remains to be discovered about the basic concept of cholesterol-lowering therapy. In this, MER/29 is comparable to the well-accepted use of antihypertensive agents: we know they lower blood pressure, but we cannot prove that lowering blood pressure will also lower morbidity or mortality. Yet few physicians hesitate to use these agents. The possible good is too great to ignore.

So it is with MER/29. No one can yet be certain that sustained, effective lowering of total body sterols will prevent or alter atherosclerosis. But the current evidence strongly supports this concept.

Perhaps that's why a growing number of physicians are now prescribing MER/29. They wish to assure their hypercholesterolemic, coronary artery disease, and atherosclerotic patients this reasonable hope.

It is a decision facing every physician.

Complete bibliography and prescription information on request.



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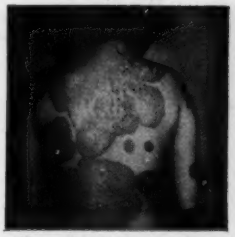

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1. Welsh, A. L.: Report, Conference on the Management of Chronic Dermatoses, University of Cincinnati College of Medicine, Cincinnati, Ohio, November 4-5, 1959.
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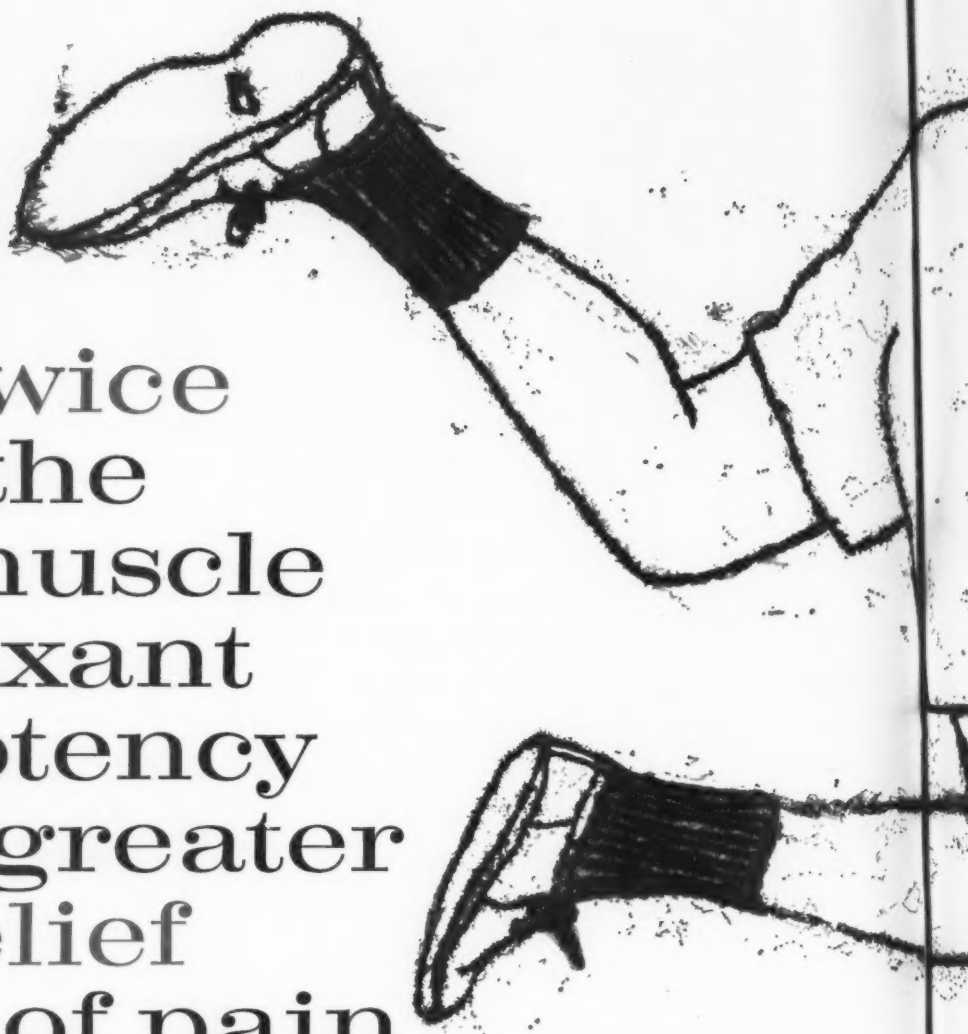

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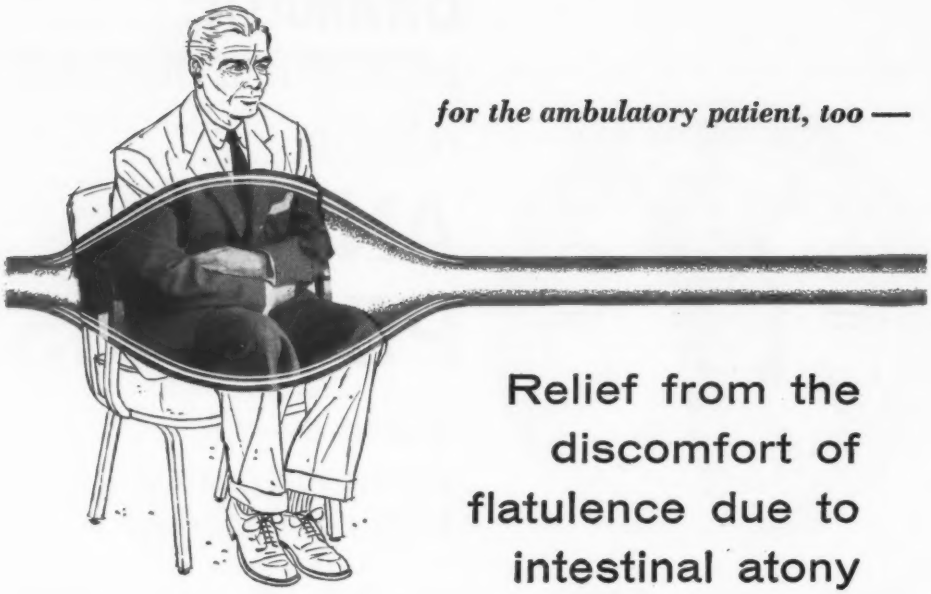

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The successful use of parenteral ILOPAN, in thousands of hospitals, for prevention and relief of post-surgical retention of flatus and feces, has brought demands for similarly effective medication for ambulatory patients — those suffering from intestinal atonia and/or gas retention, as such or as complications of geriatric problems, gastric hyperacidity, gastritis, pregnancy, irritable colon, ureteroenterostomy, regional ileitis, splenic flexure syndrome, infectious hepatitis, cholecystitis.

To ILOPAN (brand of d-pantothenyl alcohol) which aids formation of coenzyme A (essential to acetylation of choline) has been added Choline, the parent substance of acetylcholine (necessary for gastrointestinal tonus). *Effectiveness? — 90% in three independent clinical evaluations of patients of all ages from 20 to 80! And safe.*

COMPOSITION: Each tablet contains Ilopan (brand of d-pantothenyl alcohol) 50 mg., choline bitartrate 25 mg.

INDICATIONS: Gas retention in the atonic gastrointestinal tract of ambulatory patients.

DOSAGE: Two tablets three times daily. Three tablets three times daily in severe cases.

HOW SUPPLIED: Bottles of 100 and 500.

WARREN-TEED PHARMACEUTICALS

THE WARREN-TEED PRODUCTS COMPANY

COLUMBUS 8, OHIO

Dallas Chattanooga Los Angeles Portland

A-170

iron utilization improves the picture

In the "secondary" anemias due to chronic disease or infection, iron alone is often ineffective since its utilization is impeded by depressed bone marrow activity. However, RONCOVITE®-MF (cobalt-iron) has proved notably effective in these iron-refractory anemias[1,2] because of the unique marrow-activating effect of cobalt-created erythropoietin, the hormone which controls the rate of erythropoiesis. Thus, RONCOVITE-MF improves iron utilization and produces rapid increases in hemoglobin and red blood cell formation.[3,4]

Each tablet contains: Cobalt chloride, 15 mg. (cobalt as Co, 3.7 mg.) and ferrous sulfate exsiccated, 100 mg.

RONCOVITE®-mf

(1) Weinsaft, P. P., and Bernstein, L. H. T.: Am. J. M. Sc. 230:264, 1955. (2) Gosselin, G., and Long, L. A.: Appl. Therap. 2:453, 1960. (3) Rohn, R. J.; Bond, W. H., and Klotz, L. J.: Journal-Lancet 73:317, 1953. (4) Center, W. M.: Clin. Med. 7:713, 1960. V-014-61 119-R

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Each 0.6 cc. contains:

VITAMINS: A...3000 USP Units; D...400 USP Units; C...60 mg.; B_1...1 mg.; B_2...1.2 mg.; B_6...0.5 mg.; B_{12}...1 mcg. and Niacinamide...10 mg.

DOSAGE: Infants 0.3 cc. on the tongue or in the formula. Older Children 0.6 cc.

AVAILABLE: Bottles of 30 cc. at all pharmacies.

When formula days are over

and for all the other members of the family

pleasant tasting MULVIDREN® in SOFTAB® form

melts in the mouth—no water needed—the multivitamin EVERYBODY likes

EACH TABLET CONTAINS:

VITAMINS: A...5000 USP Units; D...1000 USP Units; C...75 mg.; B_1...2 mg.; B_2...2 mg.; B_6...1.2 mg.; B_{12} (as Ionex-12*), 3 mcg.; d-Calcium Pantothenate...3 mg.; Niacinamide...10 mg.

DOSAGE: 1 tablet daily or more as directed by physician.

AVAILABLE: Bottles of 50 & 100 tablets at all pharmacies.

*Stuart Ionex-12 increases Vitamin B_{12} absorption.

THE STUART COMPANY **Stuart** PASADENA, CALIFORNIA

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ADVERTISERS' INDEX

Abbott Laboratories, A-58, 59
American Ferment Div., Breon Laboratories, Inc., A-78
American Meat Institute, A-16
American Osteopathic Association, A-57, 66, 89, 133, 135, 142
Ames Company, Inc., A-154
Amfre-Grant, Inc., A-169
A-T Surgical Co., A-142

Baker Laboratories, Inc., The, A-48
Baxter, Don, Inc., A-33, 34
Becton, Dickinson & Co., A-73
Borcherdt Company, A-173
Bristol Laboratories, Inc., A-106, 107, 108, 109, 110, 111
Bristol-Myers Co., A-87
Burroughs Wellcome & Co., A-22, 56

Camp, S. H., & Co., A-134
Carnation Company, A-10, 120
Ciba Pharmaceutical Products, A-42, Cover 4
Columbus Pharmacal Co., The, A-32
Colwell Company, The, A-153
Cutter Laboratories, A-68

Davol Rubber Co., A-69
Desitin Chemical Co., A-126
Dome Chemical, Inc., A-19

Eaton Laboratories, A-98, 147
Endo Laboratories, A-164

Florida Citrus Commission, A-82

Gebauer Chemical Co., A-142
Geigy Pharmaceuticals, A-49
Greer, John F., Co., A-153

Hollister, Inc., A-84

Lakeside Laboratories, Inc., A-101
Lea & Febiger, A-150
Lederle Laboratories, A-8, 9, 43, 67, 113, 115, 117
Leeming, Thos., & Co., Inc., A-75
Lilly, Eli & Co., A-72, 128, 129
Lippincott, J. B., Co., A-168
Lloyd Bros., Inc., A-171
Lorvic Corp., The, A-80

Maltbie Labs., Div., Wallace & Tiernan, Inc., A-36, 95
Massengill, S. E., Co., A-60, 61
McNeil Laboratories, Inc., A-23, 35, 54, 55, 166, 167
Mead Johnson & Co., A-11, 62, 63, 83, 96, 97, 116
Merck Sharp & Dohme, A-26, 37, 38, 39, 50, 85, 102, 127, 144, 145
Merrell, The Wm., S., Company, A-162, 163
Metabolic Products Corp., A-155

Organon, Inc., A-40
Ortho Pharmaceutical Corp., A-99

Parke, Davis & Co., A-81, 121
Pfizer Laboratories, A-156, 157
Picker X-Ray Corp., A-1
Pitman-Moore Co., A-90, 160, 161
Purdue Frederick, The, Co., Cover 2

Reed & Carnrick, A-165
Riker Laboratories, Cover 3
Robins, A. H., Co., Inc., A-130
Roche Laboratories, A-18, 76, 77, 151
Rorer, William H., Inc., A-31, 152
Roussel Corp., A-86, 103

Sandoz Pharmaceuticals, A-88, 118, 119, 148, 149, 174
Schering Corp., A-3, 27, 44, 45, 136, 137
Schmid, Julius, Inc., A-14
Searle, G. D., & Co., A-46, 47, 71
Sherman Laboratories, A-100
Shield Laboratories, A-94
Smith Kline & French Labs., A-20, 21
Squibb, E. R., & Sons, A-146, 158
Strasenburgh Laboratories, A-12, 13
Stuart Co., The, A-6, 25, 51, 52, 132, 172

Taylor Instruments Co., A-138

U.S. Vitamin & Pharmaceutical Corp., A-104, 105, 122, 123
Upjohn Company, The, A-4, 15, 143

Wallace Laboratories, A-28, 29, 41, 53, 64, 91, 114
Warner-Chilcott Laboratories, Div., A-5, 17, 74, 92, 93
Warren-Teed Products Co., A-170
White Laboratories, A-24, 70, 112, 131, 139, 140, 141, 159
Wilco Laboratories, Inc., A-79
Winthrop Laboratories, A-7, 65, 124, 125
Wynn Pharmacal Corporation, A-30

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(MALTSUPEX) ®
POWDER LIQUID

is gentle, safe, sure, dietary

It's available in two forms, liquid and powder, but most adults prefer the mild tasting powder. It dissolves instantly in milk, water or juices. It promotes aciduric flora in the lower bowel which helps restore normal function. Long term treatment produces no side effects. Diabetic patients should allow for 60 calories for each tablespoonful.

Hootnick(1) reports, "Stools became soft in all patients and, within one week, bowel evacuations were accomplished with ease. Most patients liked the taste of the product, and the majority of them reported a feeling of well-being."

Cass and Frederik(2) also found that "Malt Soup Extract produced soft, easily evacuated stools without any side effects in constipated elderly patients."

Marshall(3) found it "a simple but highly effective treatment for chronic constipation in patients of all ages."

Dose: 2 tablespoonfuls twice a day. Available, liquid and powder, 8 ounce and 16 ounce bottles, at pharmacies.

Send for clinical samples

(1) Hootnick, H. L.: Jnl. Amer. Ger. Soc., 4:1021-1030, 1956. (2) Cass, L. J. and Frederik, W. S.: Jnl. Lancet, 73: 414-418, 1953. (3) Marshall, W.: So. Dak. J. Med. & Pharm. 8:151:153, 1955.

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***Soothes*... Burning Urination**

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Urolitia is especially useful for elderly patients with residual urine due to cystocele or enlarged prostate, in whom permanent sterilization of the urine cannot be expected.

CONTAINS NO DYES

Urolitia—each tablespoonful contains:

Methenamine	20 gr.
Lithium Benzoate	5 gr.
Sodium Benzoate	2 gr.

In a soothing, demulcent menstruum of Triticum and Zea.

Dose: 1 Tbs. in ½ cup warm water ½ hr. a.c. and h.s. Decrease dose after second day.

Supplied: Bottles of 8 fl. oz.

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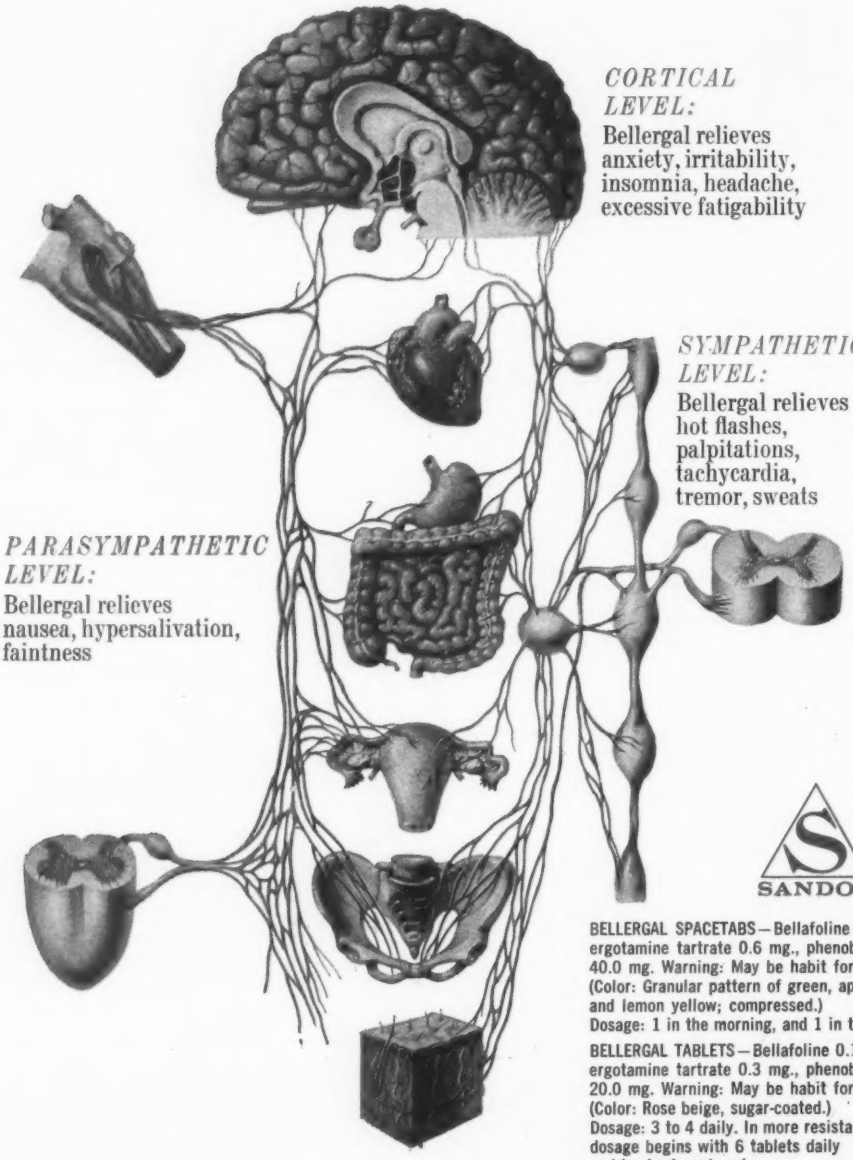
stabilizes the entire autonomic nervous system

(*without disturbing endocrine balance*)



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BELLERGAL SPACETABS—Bellafoline 0.2 mg., ergotamine tartrate 0.6 mg., phenobarbital 40.0 mg. Warning: May be habit forming. (Color: Granular pattern of green, apricot and lemon yellow; compressed.)
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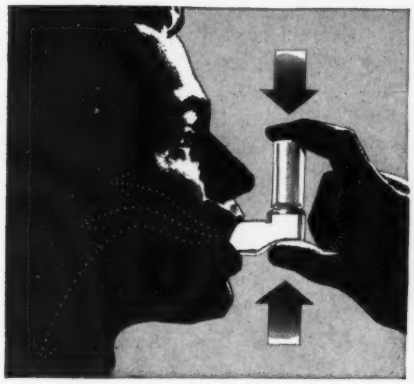

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